MEDICO-CHIRURGIC

TRANSACTIONS

CONTENTS

269489

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PRESIDENTS OF THE SOCIETY FROM ITS FORMATION AS "THE MEDICO-CHIRURGICAL SOCIETY," 1805

ELECTED

1805　WILLIAM SAUNDERS, M.D.
1808　MATTHEW BAILLIE, M.D.
1810　SIR HENRY HALFORD, Bart., M.D., G.C.H.
1813　SIR GILBERT BLANE, Bart., M.D.
1815　HENRY CLINE
1817　WILLIAM BABINGTON, M.D.
1819　SIR ASTLEY PASTON COOPER, Bart., K.C.H.
1821　JOHN COOKE, M.D.
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1825　GEORGE BIRKBECK, M.D.
1827　BENJAMIN TRAVERS
1829　PETER MARK ROGET, M.D.
1831　SIR WILLIAM LAWRENCE, Bart.
1833　JOHN ELLIOTSON, M.D. (First President of the Society after its Incorporation as the Royal Medical and Chirurgical Society of London in 1834).
1835　HENRY EARLE
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1861　BENJAMIN GUY BABINGTON, M.D.
1863　RICHARD PARTRIDGE
1865　SIR JAMES ALDERSON, M.D.
1867　SAMUEL SOLLY
1869　SIR GEORGE BURROWS, Bart., M.D.
1871　THOMAS BLIZARD CURLING
1873　CHARLES JAMES BLASIUS WILLIAMS, M.D.
1875　SIR JAMES PAGET, Bart.
1877　CHARLES WEST, M.D.
1879　JOHN ERIC ERICHSEN
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1890　TIMOTHY HOLMES
1892　SIR ANDREW CLARK, Bart., M.D.
　　　(Sir Andrew Clark died 6th November, 1893, and Dr. W. S. Church, Senior [Medical] Vice-President, officiated as President until the following 1st March, 1894.)
1894　JONATHAN HUTCHINSON
1896　WILLIAM HOWSHIP DICKINSON, M.D.
1898　THOMAS BRYANT

HONORARY FELLOWS

(Limited to Twelve.)

Elected

1887 FOSTER, SIR MICHAEL, K.C.B., M.D., LL.D., F.R.S., Professor of Physiology in the University of Cambridge.

1883 FRANKLAND, SIR EDWARD, K.C.B., M.D., D.C.L., Ph.D., F.R.S., Associate Member of the Academy of Sciences of France ; The Yews, Reigate Hill, Reigate.

1868 HOOKER, SIR JOSEPH DALTON, M.D., C.B., G.C.S.I., D.C.L., LL.D., F.R.S., Corresponding Member of the Academy of Sciences of France ; The Camp, Sunningdale.

1896 KELVIN, LORD, G.C.V.O., F.R.S., Pres.R.S.E., D.C.L., LL.D., &c., Glasgow.

1878 LUBBOCK, The Right Hon. SIR JOHN, Bart., M.P., D.C.L. LL.D., F.R.S., High Elms, Farnborough, Kent, R.S.O.

1873 STOKES, SIR GEORGE GABRIEL, Bart., M.A., D.C.L., LL.D., Sc.D., F.R.S., Lucasian Professor of Mathematics in the University of Cambridge ; Lensfield Cottage, Cambridge.

1887 TURNER, SIR WILLIAM, M.B., D.C.L., LL.D., F.R.S., Professor of Anatomy in the University of Edinburgh ; 6, Eton Terrace, Edinburgh.

FOREIGN HONORARY FELLOWS

Elected

1878   BACCELLI, GUIDO, M.D., Rome.

1896   VON BERGMANN, ERNST, Berlin.

1887   BILLINGS, JOHN S., M.D., D.C.L.Oxon., New York.

1896   CZERNY, VINCENT, M.D., Heidelberg.

1896   ERB, WILHELM, M.D., Professor of Clinical Medicine, Heidelberg.

1887   VON ESMARCH, His Excellency FRIEDRICH, M.D., Kiel.

1896   FOURNIER, ALFRED, M.D., Paris.

1896   GERHARDT, CARL, M.D., Berlin.

1896   KOCH, ROBERT, M.D., Berlin.

1896   KOCHER, THEODORE, M.D., Berne.

1868   KÖLLIKER, ALBERT, Würzburg.

1896   LAVERAN, A., M.D., Paris.

1896   MARIE, PIERRE, M.D., Paris.

1896   MITCHELL, SAMUEL WEIR, M.D., Philadelphia.

1896   MIRZA-ALI, M.D., Teheran.

1856   VIRCHOW, RUDOLF, M.D., LL.D., Berlin.

FELLOWS

ROYAL MEDICAL AND CHIRURGICAL SOC

OF LONDON

EXPLANATION OF THE ABBREVIATIONS

P.—President.   C.—Member of Council.
V.P.—Vice-President.   *Sci. Com.*—Member of a Scientific Con
T.—Treasurer.   *Ho. Com.*—Member of House Commit
L.—Hon. Librarian.   *Lib. Com.*—Member of Library Commi
S.—Hon. Secretary.   *Bldg. Com.*—Member of Building Com
                Dis. Com.—Member of Discussions Committee.

The abbreviations *Trans.* and *Pro.*, followed by figures, show the nur
Papers which have been contributed to the *Transactions* o:
ceedings by the Fellow whose name they follow. *Referee, Sci. Co?*
Com., Bldg. Com., Ho. Com., and *Dis. Com.,* with the dates of off
attached to the names of those who have served as Referees of
and on the Committees of the Society.

Names printed in this **type** are of those Fellows who have paid the (
position Fee in lieu of further annual subscriptions.

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position Fee entitling them to receive the *Transactions.*

RESIDENT FELLOWS

[N.B.—Fellows are reminded that they are, themselves, responsible f(
correctness of the descriptions in the following lists, and
particularly requested that any change of Title, Appointme
Residence may be communicated to the Hon. Secretaries befo:
1st of July in each year.]

Elected

1898   AARONS, S. JERVOIS, M.D., 15, Devonshire place, :
           land place.

1877   **Abercrombie, JOHN, M.D.,** Physician to, and Lectur(
           Forensic Medicine at, Charing Cross Hospital
           Upper Wimpole street, Cavendish square.   C. 18
           Referee, 1898.   *Trans.* 2.

Elected

1885   ABRAHAM, PHINEAS S., M.A., M.D., Dermatologist to the West London Hospital, Assistant Surgeon to Hospital for Diseases of the Skin, Blackfriars; 2, Henrietta street, Cavendish square.

1885   ACLAND, THEODORE DYKE, M.D., Physician to St. Thomas's Hospital, and Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 74, Brook street, Grosvenor square.

1852   **Adams,** WILLIAM, Consulting Surgeon to the Great Northern Central Hospital, the National Hospital for the Paralysed and Epileptic, and the National Orthopædic Hospital; 7, Loudoun road, St. John's Wood. C. 1873-4. *Trans.* 3.

1879   ALLCHIN, WILLIAM HENRY, M.D., F.R.S. Ed., Senior Physician to the Westminster Hospital; 5, Chandos street, Cavendish square. C. 1898-9. *Referee,* 1897.

1890   ALLINGHAM, HERBERT WILLIAM, Surgeon to the Great Northern Hospital; Assistant Surgeon to St. George's Hospital; 25, Grosvenor street, Grosvenor square.

1863   **Althaus,** JULIUS, M.D., Consulting Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 26, Queen Anne street, Cavendish square. *Trans.* 2.

1888   ANDERSON, JOHN, M.D., C.I.E., Physician to the Seamen's Hospital, Greenwich; Lecturer on Tropical Medicine at St. Mary's Hospital Medical School; 9, Harley street, Cavendish square.

1890   ANDERSON, WILLIAM, Surgeon to St. Thomas's Hospital; Professor of Anatomy to the Royal Academy of Arts; 5, Cavendish square. *Lib. Com.* 1896-8.

1891   ANDREWES, FREDERICK WILLIAM, M.D., Highwood, Hampstead Lane, Highgate.

1893   BAILEY, ROBERT COZENS, M.S., 21, Welbeck street, Cavendish square.

Elected

1891 BAKER, CHARLES ERNEST, M.B., 5, Gledhow gardens, South Kensington.

1899 BAKER, OSWALD, 57, Welbeck street, Cavendish square.

1887 BALL, JAMES BARRY, M.D., Physician to the West London Hospital; 12, Upper Wimpole street, Cavendish square.

1885 BALLANCE, CHARLES ALFRED, M.S., Assistant Surgeon to St. Thomas's Hospital and to the Hospital for Sick Children, Great Ormond street; Surgeon to the National Hospital for the Paralysed and Epileptic, Queen square; 106, Harley street, Cavendish square. *Trans.* 4.

1879 BARKER, ARTHUR EDWARD JAMES, Professor of the Principles and Practice of Surgery and Professor of Clinical Surgery at University College, and Surgeon to University College Hospital, London; 87, Harley street, Cavendish square. C. 1895-7. *Referee,* 1897—. *Trans.* 7.

1876 **Barlow,** THOMAS, M.D., B.S., *Hon. Secretary, Trustee for Debenture - holders*; Physician-in-Ordinary to H. M.'s Household; Physician to University College Hospital; 10, Wimpole street, Cavendish square. C. 1892. S. 1899—. *Referee,* 1896-9. *Trans.* 2.

1893 BARRETT, HOWARD, 49, Gordon square.

1880 BARROW, A. BOYCE, Surgeon to King's College Hospital; 37, Wimpole street, Cavendish square.

1896 BARTON, JAMES KINGSTON, 2, Courtfield road, Gloucester road, South Kensington.

1859 BARWELL, RICHARD, Consulting Surgeon to the Charing Cross Hospital; 55, Wimpole street. C. 1876-77. V.P. 1883-4. *Referee,* 1868-75, 1879-82. *Trans.* 12. *Pro.* 1.

Elected

1868   **Bastian,** Henry Charlton, M.A., M.D., F.R.S., Emeritus Professor of the Principles and Practice of Medicine and of Clinical Medicine in University College, London ; Consulting Physician to University College Hospital and Physician to the National Hospital for the Paralysed and Epileptic ; 8A, Manchester square. C. 1885. *Referee,* 1886-96. *Trans.* 3.

1890   Bateman, William A. F., Bridge House, Richmond, Surrey.

1891   Batten, Frederick E., M.D., B.C., 124, Harley street.

1875   Beach, Fletcher, M.B., Winchester House, Kingston Hill [64, Welbeck street].

1883   Beale, Edwin Clifford, M.A., M.B., Physician to the City of London Hospital for Diseases of the Chest, and Physician to the Great Northern Central Hospital ; 23, Upper Berkeley street.

1862   Beale, Lionel Smith, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital ; 61, Grosvenor street. C. 1876-7. *Referee,* 1873-5. *Trans.* 1.

1897   Beddard, A. P., M.B., 44, Seymour street.

1880   Beevor, Charles Edward, M.D., Physician for Out-patients to the National Hospital for the Paralysed and Epileptic, and to the Great Northern Hospital ; 33, Harley street, Cavendish square. *Referee,* 1896—. *Trans.* 1

1883   Bennett, Storer, Dental Surgeon to, and Lecturer on Dental Surgery at the Middlesex Hospital ; 17, George street, Hanover square.

1877   Bennett, William Henry, Surgeon to St. George's Hospital ; 1, Chesterfield street, Mayfair. C. 1893-4. *Referee,* 1892-93, 1899. *Trans.* 4.

Elected

1897 BERKELEY, COMYNS, M.B., B.C., Physician to Out-Patients, Chelsea Hospital for Women ; 53, Wimpole street.

1845 **Berry,** EDWARD UNWIN, 17, Sherriff road, West Hampstead.

1885 BERRY, JAMES, B.S., Surgeon to the Royal Free Hospital, and Lecturer on Surgery at the London School of Medicine for Women ; Demonstrator of Practical Surgery, St. Bartholomew's Hospital ; 60, Welbeck street, Cavendish square.

1893 BIDWELL, LEONARD A., Senior Assistant Surgeon to the West London Hospital ; 59, Wimpole street.

1856 **Bird,** WILLIAM, Consulting Surgeon to the West London Hospital ; Bute House, Hammersmith.

1851 **Birkett,** JOHN, F.L.S., Consulting Surgeon to Guy's Hospital ; Corresponding Member of the Société de Chirurgie of Paris ; 1, Sussex gardens. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. V.P. 1879-80. *Referee,* 1851-5, 1866, 1869. *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.

1897 BLACKER, G. F., M.D., 11, Wimpole street, Cavendish square.

1865 **Blandford,** GEORGE FIELDING, M.D., Lecturer on Psychological Medicine at St. George's Hospital ; 48, Wimpole street, Cavendish square. C. 1883-4. V.P. 1898—.

1891 BOKENHAM, THOMAS JESSOPP, 10, Devonshire street, Portland place.

1882 BOWLBY, ANTHONY ALFRED, Assistant Surgeon to St. Bartholomew's Hospital ; 24, Manchester square. *Trans.* 8.

1870 *Bowles,* ROBERT LEAMON, M.D., 16, Upper Brook street, Grosvenor square. C. 1897-9. *Sci. Com.* 1896—. *Trans.* 2.

1886 BOXALL, ROBERT, M.D., Assistant Obstetric Physician to, and Lecturer on Practical Midwifery at, the Middlesex Hospital ; 40, Portland place.

Elected

1884   BOYD, STANLEY, M.B., Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital; Surgeon to the Paddington Green Children's Hospital; Consulting Surgeon to the New Hospital for Women ; 134, Harley street, Cavendish square. *Referee*, 1895—. *Trans.* 1.

1890   BRADFORD, JOHN ROSE, M.D., D.Sc., F.R.S., Physician to University College Hospital; 60, Wimpole street. *Referee*, 1899. *Trans.* 1.

1897   BRAILEY, WILLIAM ARTHUR, M.D., 11, Old Burlington street.

1890   BRINTON, ROLAND DANVERS, M.D., 8, Queen's Gate terrace.

1898   **Broadbent, J. F. H., M.D., 35, Seymour street.**

1868   **Broadbent, SIR WILLIAM HENRY, Bart., M.D., F.R.S., LL.D., Physician in Ordinary to H.R.H. the Prince of Wales ; Physician-Extraordinary to Her Majesty the Queen ; Consulting Physician to St. Mary's Hospital ; Consulting Physician to the London Fever Hospital ; 84, Brook street, Grosvenor square. C. 1885. *Referee*, 1881-4, 1891-7. *Trans.* 5.**

1851   **Brodhurst, BERNARD EDWARD, Surgeon to the Royal Orthopædic Hospital and to the Royal Hospital for Incurables; Consulting Surgeon, Belgrave Hospital for Children ; Corresponding Member, Société de Chirurgie, Paris, and of the Academy of Sciences, Rome ; 21, Portland place. C. 1868-9. *Lib. Com.* 1862-3. *Trans.* 2. *Pro.* 1.**

1872   BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 8, Chesterfield street, Mayfair. *Trans.* 1.

1880   BROWNE, JAMES WILLIAM, M.B., 37, Holland Park avenue.

Elected

1881 BROWNE, OSWALD AUCHINLECK, M.A., M.D., Physician to the Royal Hospital for Diseases of the Chest and to the Metropolitan Hospital; 7, Upper Wimpole street.

1874 BRUCE, JOHN MITCHELL, M.D., Physician to, and Lecturer on Medicine at, the Charing Cross Hospital; Consulting Physician to the Hospital for Consumption, Brompton; 23, Harley street. C. 1892, 1897-9. S. 1893-5. *Sci. Com.* 1889—. *Ho. Com.* 1898-9. *Referee*, 1886-91. *Lib. Com.* 1888-91. *Trans.* 3.

1898 BRUCE, SAMUEL NOBLE, 15, Queensborough terrace, Hyde Park.

1871 BRUNTON, THOMAS LAUDER, M.D., D.Sc., LL.D., F.R.S., Physician to, and Lecturer on Pharmacology and Therapeutics at, St. Bartholomew's Hospital; 10, Stratford place, Oxford street. C. 1888-9. *Referee*, 1880-87. *Lib. Com.* 1882-7. *Trans.* 2.

1898 BRYANT, J. H., M.D., Assistant Physician to Guy's Hospital; 8, St. Thomas's street, London bridge.

1860 **Bryant**, THOMAS, M.Ch., *President*, Surgeon-Extraordinary to H.M. the Queen; Consulting Surgeon to Guy's Hospital; Member of the Société de Chirurgie, Paris; 65, Grosvenor street, Grosvenor square. C. 1873-4. V.P. 1885-6. *Sci. Com.* 1863. *Referee*, 1882-4. *Lib. Com.* 1868-71. *Trans.* 15. *Pro.* 1.

1889 BULL, WILLIAM CHARLES, M.B., Aural Surgeon to, and Lecturer on Aural Surgery at, St. George's Hospital; 5, Clarges street, Piccadilly.

1893 BURGHARD, FRÉDÉRIC FRANÇOIS, M.D., M.S., Surgeon to King's College Hospital and Paddington Green Children's Hospital; 86, Harley street, Cavendish square.

1885 BUTLER-SMYTHE, ALBERT CHARLES, Senior Out-Patient Surgeon, Samaritan Free Hospital for Women and Children, Soho; Senior Surgeon to the Grosvenor Hospital for Women and Children; 76, Brook street, Grosvenor square.

Elected

1873   Butlin, Henry Trentham, D.C.L., Surgeon to St.
        Bartholomew's Hospital; 82, Harley street, Cavendish
        square. C. 1887-8. *Referee*, 1893—. *Trans.* 4. *Pro.* 1.

1896   Buttar, Charles, M.D., 10, Kensington gardens square,
        Bayswater. *Pro.* 1.

1883   Buxton, Dudley Wilmot, M.D., B.S., Administrator, and
        Teacher of the Use, of Anæsthetics, in University College
        Hospital; Consulting Anæsthetist to the National Hos-
        pital for the Paralysed and Epileptic, Queen square,
        and Anæsthetist to the London Dental Hospital; 82,
        Mortimer street, Cavendish square.

1868   Buzzard, Thomas, M.D., Physician to the National Hos-
        pital for the Paralysed and Epileptic; 74, Grosvenor
        street, Grosvenor square. C. 1885-6. *Referee*, 1887—.

1885   Cahill, John, M.D., Surgeon to the Hospital of St.
        John and St. Elizabeth; 12, Seville street, Lowndes
        square.

1893   Caley, Henry Albert, M.D., Physician in charge of
        Out-patients, Lecturer in Materia Medica and Thera-
        peutics, and Senior Medical Tutor, St. Mary's Hos-
        pital; 24, Upper Berkeley street, Portman square.

1887   Calvert, James, M.D., The Warden's House, St. Bartho-
        lomew's Hospital. *Trans.* 1.

1897   Cantlie, James, M.B., 46, Devonshire street.

1888   Carless, Albert, M.S., Assistant Surgeon to King's
        College Hospital; 10, Welbeck street.

1896   Carr, J. Walter, M.D., Assistant Physician to the Royal
        Free Hospital; 19, Cavendish place. *Trans.* 1.

1875   Carter, Charles Henry, M.D., Physician to the Hospital
        for Women, Soho square; 45, Great Cumberland place,
        Hyde Park.

1898   Carter, H. Ronald, 11, Leonard place, Kensington.

1853   Carter, Robert Brudenell, Consulting Ophthalmic
        Surgeon to St. George's Hospital; 31, Harley street,
        Cavendish square, and Kenilworth, Clapham common.
        Trans. 1.

Elected

· 1888 CAUTLEY, EDMUND, M.D., B.C., 15, Upper Brook street. *Trans.* 1.

1871 CAYLEY, WILLIAM, M.D., Physician to the Middlesex Hospital, Consulting Physician to the London Fever Hospital, and to the North-Eastern Hospital for Children; 27, Wimpole street, Cavendish square. C. 1888. *Referee*, 1886-7, 1899, *Lib. Com.* 1886-7. *Trans.* 2.

1879 CHAMPNEYS, FRANCIS HENRY, M.D., Physician-Accoucheur and Lecturer on Obstetric Medicine at St. Bartholomew's Hospital; 42, Upper Brook street, Grosvenor square. C. 1898—. *Referee*, 1891-8. *Lib. Com.* 1885-8. *Trans.* 8.

1868 **Cheadle**, WALTER BUTLER, M.D., *Trustee;* Physician to and Lecturer on Clinical Medicine at, St. Mary's Hospital; Consulting Physician to the Hospital for Sick Children; 19, Portman street, Portman square. S. 1886-8. C. 1890-91. *Sci. Com.* 1889-95. *Bldg. Com.* 1889-92. *Referee*, 1885. *Trans.* 1.

1879 CHEYNE, WILLIAM WATSON, M.B., F.R.S., Surgeon to King's College Hospital, and Professor of Surgery in King's College, London; 75, Harley street, Cavendish square. C. 1897-9. *Referee*, 1894-7. *Lib. Com.* 1886-8, 1891-6. *Trans.* 1.

1890 CHILDS, CHRISTOPHER, M.D., 10, Manchester square.

1896 CHRISTOPHERSON, JOHN BRIAN, M.D., B.C., Assistant Demonstrator of Anatomy at St. Bartholomew's Hospital; 5, Staple inn, Holborn.

1866 **Church**, WILLIAM SELBY, M.D., *Hon. Treasurer*, President of the Royal College of Physicians of London, Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. C. 1885-6. V.P. 1892-4. T. 1894—. *Referee*, 1874-81. *Ho. Com.* 1898—.

1879 CLARK, ANDREW, Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 71, Harley street, Cavendish square.

Elected

1882   CLARKE, ERNEST, M.D., B.S., Surgeon to the Central
       London Ophthalmic Hospital; Ophthalmic Surgeon to
       the Miller Hospital; 3, Chandos street, Cavendish
       square.

1890   CLARKE, JAMES JACKSON, M.B., Assistant Surgeon to the
       North-West London and City Orthopædic Hospitals;
       9, Old Cavendish street, Cavendish square.

1848   Clarke, JOHN, M.D., 42, Hertford street, Mayfair.
       C. 1866.

1881   CLARKE, W. BRUCE, M.B., Assistant Surgeon to, and
       Lecturer on Anatomy at, St. Bartholomew's Hospital;
       Surgeon to the West London Hospital; 51, Harley
       street, Cavendish square.   C. 1899—.   *Trans.* 1.

1879   Clutton, HENRY HUGH, M.B., M.C., Senior Surgeon to,
       and Lecturer on Surgery at, St. Thomas's Hospital;
       2, Portland place.   C. 1897-9.   *Dis. Com.* 1897-8.
       Referee, 1896-7.   *Trans.* 3.

1888   COCK, FREDERICK WILLIAM, M.D., 1, Porchester Houses,
       Porchester square.

1897   COLMAN, W. S., M.D., Assistant Physician to St. Thomas's
       Hospital; 22, Wimpole street.

1865   COOPER, ALFRED, Surgeon in Ordinary to H.R.H. the
       Duke of Saxe Coburg-Gotha; Consulting Surgeon
       to the West London Hospital and to St. Mark's Hos-
       pital; 9, Henrietta street, Cavendish square.

1898   CORFIELD, W. H., M.D., Professor of Hygiene and Public
       Health at University College, London; Medical
       Officer of Health for St. George's, Hanover square;
       19, Savile row, and Whindown, Bexhill, Sussex.

1889   COSENS, CHARLES HENRY, 49, Oxford terrace, Hyde Park.

1860   Couper, JOHN, Consulting Surgeon to the Royal London
       Ophthalmic Hospital and to the London Hospital;
       80, Grosvenor street.   C. 1876.   *Referee*, 1882-3.

Elected

1877 COUPLAND, SIDNEY, M.D., Commissioner in Lunacy; late Physician to, and Lecturer on Medicine at, the Middlesex Hospital; 16, Queen Anne street, Cavendish square. C. 1893-4. *Referee*, 1892-3. *Ho. Com.* 1895-8.

1862 **Cowell**, GEORGE, Consulting Surgeon to the Westminster Hospital and to the Royal Westminster Ophthalmic Hospital; 19, Harley street, Cavendish square. C. 1882-3.

1897 CRAWFURD, RAYMOND H. PAYNE, M.D., 71, Harley street.

1878 CRICHTON-BROWNE, SIR JAMES, M.D., LL.D., F.R.S., Lord Chancellor's Visitor in Lunacy; 61, Carlisle place Mansions, Victoria street.

1874 CRIPPS, WILLIAM HARRISON, Surgeon to St. Bartholomew's Hospital; 2, Stratford place, Oxford street. C. 1890-91. *Trans.* 1.

1882 CROCKER, HENRY RADCLIFFE, M.D., Physician to the Skin Department, University College Hospital; 121, Harley street, Cavendish square. *Trans.* 3.

1868 CROFT, JOHN, Consulting Surgeon to St. Thomas's Hospital; 6, Mansfield street, Cavendish square. C. 1884. V.P. 1890. *Referee*, 1885-88. *Lib. Com.* 1877-8. *Trans.* 2.

1898 CROMBIE, ALEXANDER, M.D., 10, Devonshire street, Portland place.

1899 CROSSE, W. H., 45, Dover street, Piccadilly.

1890 CROWLE, THOMAS HENRY RICKARD, 56, Harley street, Cavendish square.

1888 CULLINGWORTH, CHARLES JAMES, M.D., D.C.L., Obstetric Physician and Lecturer on Midwifery and Diseases of Women to St. Thomas's Hospital; 14, Manchester square. *Referee*, 1896—.

Elected

1879   CUMBERBATCH, A. ELKIN, M.B., Aural Surgeon to St. Bartholomew's Hospital, and to the National Hospital for the Paralysed and Epileptic; 60, Portland place.

1873   CURNOW, JOHN, M.D., Professor of Clinical Medicine in King's College, London, and Physician to King's College Hospital; Senior Physician to the Seamen's Hospital; 9, Wimpole street, Cavendish square. *Referee*, 1884-97.

1898   CURRIE, A. STARK, M.D., 81, Queen's road, Finsbury park.

1886   DAKIN, WILLIAM RADFORD, M.D., Obstetric Physician to, and Lecturer in Midwifery at, St. George's Hospital, and Physician to the General Lying-in Hospital; 18, Grosvenor street, Grosvenor square.

1872   DALBY, SIR WILLIAM BARTLETT, M.B., Consulting Aural Surgeon to St. George's Hospital; 18, Savile row. C. 1896-7.   *Trans.* 3.

1891   DALTON, NORMAN, M.D., Physician to King's College Hospital; Professor of Pathological Anatomy in King's College, London; 4, Mansfield street, Cavendish square.

1896   DAUBER, JOHN HENRY, M.B., B.Ch., Assistant Physician to the Hospital for Women, Soho square; 29, Charles street, Berkeley square.

1876   DAVIES-COLLEY, J. NEVILLE C., M.C., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 36, Harley street, Cavendish square. C. 1892-3. *Referee*, 1890-91. *Trans.* 3.

1889   DEAN, HENRY PERCY, M.S., Surgeon to the London Hospital; 69, Harley street, Cavendish square.

1878   DENT, CLINTON THOMAS, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 61, Brook street. C. 1890. *Bldg. Com.* 1890-2. *Referee*, 1892—. *Trans.* 5.

Elected

1891   DE SANTI, PHILIP ROBERT WILLIAM, Assistant Surgeon and Aural Surgeon to the Westminster Hospital; 42, Harley street.

1894   DICKINSON, THOMAS VINCENT, M.D., 33, Sloane street.

1859   **Dickinson**, WILLIAM HOWSHIP, M.D., Consulting Physician to St. George's Hospital, and Consulting Physician to the Hospital for Sick Children; Honorary Fellow of Caius College, Cambridge; 9, Chesterfield street, Mayfair. P. 1896-8. C. 1874-5. V. P. 1887. *Referee*, 1869-73. 1882-6. *Sci. Com.* 1867, 1879, 1889-96. *Trans.* 16.

1891   **Dickinson**, WILLIAM LEE, M.D., Assistant Physician to St. George's Hospital and to the Hospital for Sick Children; 9, Chesterfield street, Mayfair.

1889   DODD, HENRY WORK, Surgeon to the Royal Westminster Ophthalmic Hospital; Ophthalmic Surgeon to the Royal Free Hospital and to the West-End Hospital for Nervous Diseases; 136, Harley street, Cavendish square.

1888   DONELAN, JAMES, M.B., M.C., Physician to the Italian Hospital, Queen square; 2, Upper Wimpole street, Cavendish square.

1877   DORAN, ALBAN HENRY GRIFFITHS, Surgeon to the Samaritan Free Hospital; 9, Granville place, Portman square. C. 1893-4. *Lib. Com.* 1891-3, 1899. *Referee*, 1898—. *Trans.* 3.

1867   DOUGLAS-POWELL, SIR RICHARD, Bart., M.D., Physician in Ordinary to H.M. the Queen; Physician to, and Lecturer on Medicine at, the Middlesex Hospital; Consulting Physician to the Hospital for Consumption, Brompton; 62, Wimpole street, Cavendish square. S. (Oct.) 1883-5. C. 1887-8. *Referee*, 1879-83, 1886. *Trans.* 3.

1891   DOVE, PERCY W., M.B., 80, Crouch hill.

1896   DOWNES, JOSEPH LOCKHART, M.B., C.M., 271, Romford road.

Elected

1879   DREWITT, F. G. DAWTREY, M.D., Physician to the West London Hospital; 2, Manchester square.

1893   DRYSDALE, JOHN H., M.B., 25, Welbeck street, Cavendish square.

1865   **Duckworth**, SIR DYCE, M.D., LL.D., Hon. Physician to H.R.H. the Prince of Wales; Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. C. 1883-4. *Referee* 1885-97. *Trans.* 2.

1876   DUDLEY, WILLIAM LEWIS, M.D., Senior Physician to the City Dispensary; 149, Cromwell road, South Kensington.

1871   DUKE, BENJAMIN, M.D., Windmill House, Clapham Common.

1880   DUNBAR, JAMES JOHN MACWHIRTER, M.D., Hedingham House, Clapham Common.

1884   DUNCAN, WILLIAM, M.D., Obstetric Physician to, and Lecturer on Midwifery at, the Middlesex Hospital; 6, Harley street, Cavendish square.

1887   DUNN, HUGH PERCY, Ophthalmic Surgeon to the West London Hospital; 54, Wimpole street, Cavendish square.

1898   DUNN, L. A., M.S., The College, Guy's Hospital.

1874   DURHAM, FREDERIC, M.B., Senior Surgeon to the North-West London Hospital; 82, Brook street, Grosvenor square.

1894   DURHAM, HERBERT EDWARD, M.B., 82, Brook street, Grosvenor Square. *Trans.* 2.

1868   **Eastes**, GEORGE, M.B.Lond., 35, Gloucester terrace, Hyde Park. C. 1892-3.

1838   ECCLES, ARTHUR SYMONS, M.B., C.M., 23, Hertford street, Mayfair.

1893   ECCLES, WILLIAM MCADAM, M.S., Assistant Surgeon to the West London Hospital and to the City of London Truss Society; 124, Harley street.

Elected

1891 EDDOWES, ALFRED, M.D., 28, Wimpole street.

1898 EDKINS, J. S., 4, Park hill road, Hampstead.

1898 EDMUNDS, P. J., M.B., 5, Great Marlborough street, Regent street.

1883 EDMUNDS, WALTER, M.C., 75, Lambeth Palace road, Albert Embankment. *Trans.* 3.

1884 EDWARDS, FREDERICK SWINFORD, Surgeon to the West London Hospital, and to St. Peter's Hospital ; Senior Assistant Surgeon to St. Mark's Hospital ; 55, Harley street, Cavendish square.

1898 EVANS, WILLMOTT, 13, Taviton street, Gordon square.

1879 EVE, FREDERIC S., Surgeon to the London Hospital ; Surgeon to the Evelina Hospital for Sick Children ; 125, Harley street, Cavendish square. C. 1897-9. *Trans.* 4.

1877 EWART, WILLIAM, M.D., Physician to St. George's Hospital and to the Belgrave Hospital for Children ; 33, Curzon street, Mayfair. C. 1895-7. *Lib. Com.* 1897—. *Sci. Com.* 1889—. *Trans.* 1. *Pro.* 1.

1872 FAYRER, SIR JOSEPH, Bart., K.C.S.I., LL.D., M.D., F.R.S., Surgeon-General ; Honorary Physician to H.M. the Queen, (Military) to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh ; late Physician to the Secretary of State for India in Council, and President of the Medical Board at the India Office ; 16, Devonshire street, Portland place. C. 1888. *Referee,* 1881-7.

1898 FENWICK, E. HURRY, Surgeon to the London Hospital and to St. Peter's Hospital ; 14, Savile row.

1863 **Fenwick**, SAMUEL, M.D., Consulting Physician to the London Hospital ; 29, Harley street, Cavendish square. C. 1880. *Referee,* 1882-96. *Trans.* 4.

1880 FERRIER, DAVID, M.D., LL.D., F.R.S., Professor of Neuro-pathology in King's College, London, and Physician to King's College Hospital ; Physician to the National Hospital for the Paralysed and Epileptic ; 34, Cavendish square. *Referee,* 1891-6. C. 1896-8. *Dis. Com.* 1896—. *Trans.* 2.

Elected

1889   FIELD, GEORGE P., Aural Surgeon to, and Lecturer on Aural Surgery at, St. Mary's Hospital; 34, Wimpole street, Cavendish square.

1891   FLETCHER, HERBERT MORLEY, M.D., Assistant Physician, East London Hospital for Children ; 98, Harley street, Cavendish square.

1892   FORSBROOK, WILLIAM HENRY RUSSELL, M.D., 139, Buckingham Palace road.

1883   FOWLER, JAMES KINGSTON, M.D., Physician to, and Lecturer on Medicine at, the Middlesex Hospital; Physician to the Hospital for Consumption, Brompton ; 35, Clarges street, Piccadilly.

1880   FOX, THOMAS COLCOTT, B.A., M.B., Physician for Diseases of the Skin to the Westminster Hospital, and Physician to the Skin Department of the Paddington Green Hospital for Children ; 14, Harley street, Cavendish square. *Trans.* 1.

1871   FRANK, PHILIP, M.D., 3, Elvaston place, South Kensington.

1896   FREYER, P. J., M.D., M.Ch., Surgeon to St. Peter's Hospital; 46, Harley street, Cavendish square. *Trans.* 1.

1898   FRIPP, A. DOWNING, M.S., Surgeon in Ordinary to H.R.H. the Prince of Wales ; Assistant Surgeon to Guy's Hospital; 19, Portland place.

1898   FROST, W. ADAMS, Ophthalmic Surgeon to St. George's Hospital, and Surgeon to Royal Westminster Ophthalmic Hospital; 30, Cavendish square.

1884   FULLER, CHARLES CHINNER, 10, St. Andrew's place, Regent's Park.

1883   FULLER, HENRY ROXBURGH, M.D., 45, Curzon street, Mayfair.

1894   FURNIVALL, PERCY, Assistant Surgeon, London Hospital; Assistant Surgeon, St. Mark's Hospital ; 39, Welbeck street, Cavendish square.

Elected

1874 **Galabin,** ALFRED LEWIS, M.D., Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital ; 49, Wimpole st., Cavendish square. C. 1892. *Referee,* 1882-91, 1896—. *Lib. Com.* 1883-4. *Trans.* 2.

1895 GALLOWAY, JAMES, M.D., Physician, Skin Department, and Joint Lecturer on Practical Medicine, Charing Cross Hospital ; 54, Harley street, Cavendish square.

1883 GALTON, JOHN CHARLES, M.A., F.L.S., 10, Upper Cheyne row, Chelsea.

1865 **Gant,** FREDERICK JAMES, Consulting Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde Park. C. 1880-81. V.P. 1897-9. *Referee,* 1886-97. *Lib. Com.* 1882-5. *Trans.* 3.

1854 **Garrod,** SIR ALFRED BARING, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital ; 10, Harley street, Cavendish square. C. 1867. V.P. 1880-81. *Referee,* 1855-65. *Trans.* 9.

1886 GARROD, ARCHIBALD EDWARD, M.D., Medical Registrar and Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; Assistant Physician to the Hospital for Sick Children, Great Ormond street ; 9, Chandos street, Cavendish square. *Sci. Com.* 1889—. *Lib. Com.* 1896—. *Trans.* 6.

1887 GAY, JOHN, 119, Upper Richmond road, Putney.

1866 **Gee,** SAMUEL JONES, M.D., *Chairman of Trustees for Debenture-holders;* Physician to St. Bartholomew's Hospital; 31, Upper Brook street, Grosvenor square. C. 1883-4. L. (June) 1887-99. V.P. 1899—. *Sci. Com.* 1879. *Bldg. Com.* 1889-92. *Referee,* 1885-7. *Lib. Com.* 1871-6. *Ho. Com.* 1898—. *Trans.* 1.

1885 GELL, HENRY WILLINGHAM, M.B., 36, Hyde Park square.

1898 GIBBES, CUTHBERT CHAPMAN, M.D., 83, Barkston gardens, Earl's Court.

1880 GIBBONS, ROBERT ALEXANDER, M.D., Physician to the Grosvenor Hospital for Women and Children; 29, Cadogan place. C. 1896-7. *Trans.* 1.

Elected

1893   GILES, ARTHUR EDWARD, M.D., B.Sc., Assistant Surgeon, Chelsea Hospital for Women ; 37, Queen Anne street.

1894   GILL, RICHARD, 17, Wigmore street.

1877   GODLEE, RICKMAN JOHN, M.S., *Hon. Librarian ;* Surgeon in Ordinary to Her Majesty's Household ; Surgeon to University College Hospital, and Professor of Clinical Surgery in University College, London ; Surgeon to the Hospital for Consumption, Brompton ; 19, Wimpole street, Cavendish square. S. 1892-4. L. 1895—. *Referee,* 1886-91. *Ho. Com.* 1898—. *Trans.* 9.

1870   **Godson,** CLEMENT, M.D., Consulting Physician to the City of London Lying-in Hospital ; 9, Grosvenor street, Grosvenor square.

1886   GOLDING-BIRD, CUTHBERT HILTON, M.B., Surgeon to, and Lecturer on Clinical Surgery at, Guy's Hospital ; 12, Queen Anne street, Cavendish square. *Trans.* 1.

1897   GOODBODY, F. W., M.D., 35, Bedford Court Mansions, Bloomsbury.

1896   GOODALL, EDWARD WILBERFORCE, M.D., B.S., Eastern Hospital, Homerton.

1883   GOODHART, JAMES FREDERIC, M.D., Physician to Guy's Hospital ; Consulting Physician to the Evelina Hospital for Sick Children ; 25, Portland place. *Lib. Com.* 1893-6.

1889   GOODSALL, DAVID HENRY, Surgeon to the Metropolitan Hospital ; Surgeon to St. Mark's Hospital ; 17, Devonshire place, Upper Wimpole street.

1895   GOSSAGE, ALFRED MILNE, M.B., 54, Upper Berkeley street.

1877   GOULD, ALFRED PEARCE, M.S., *Hon. Secretary,* Surgeon to, and Lecturer on Surgical Pathology at, the Middlesex Hospital; 10, Queen Anne street, Cavendish square. C. 1892-3. S. 1898—. *Referee,* 1895-8. *Ho. Com.* 1892-8. *Lib. Com.* 1891. *Trans.* 2.

Elected

1891 Gow, WILLIAM J., M.D., Assistant Obstetric Physician
to St. Mary's Hospital; Obstetric Physician to the
Royal Hospital for Women and Children; Physician
to Out-Patients, Queen Charlotte's Lying-in Hospital;
27, Weymouth street, Portland place.

1873 **Gowers,** SIR WILLIAM RICHARD, M.D., F.R.S., Consulting
Physician to University College Hospital; Physician
to the National Hospital for the Paralysed and Epileptic; 50, Queen Anne street, Cavendish square.
C. 1891. *Referee* 1888-90. *Lib. Com.* 1884-6. *Trans.* 7.

1892 GRANT, J. DUNDAS, M.A., M.D., 8, Upper Wimpole street,
Cavendish square.

1898 GRANVILLE, ALEXANDER, St. Bartholomew's Hospital.

1868 GREEN, T. HENRY, M.D., Physician to the Charing Cross
Hospital, and to the Hospital for Consumption,
Brompton; 74, Wimpole street, Cavendish square.
C. 1886. *Referee,* 1882-5.

1885 GRIFFITH, WALTER SPENCER ANDERSON, M.D., Assistant
Physician-Accoucheur, St. Bartholomew's Hospital;
Physician to Queen Charlotte's Lying-in Hospital;
96, Harley street, Cavendish square.

1868 GRIGG, WILLIAM CHAPMAN, M.D., Senior Physician to
Queen Charlotte's Lying-in Hospital; 27, Curzon
street, Mayfair.

1889 GUBB, ALFRED S., M.D., 29, Gower street.

1883 GUNN, ROBERT MARCUS, M.B., Surgeon to the Royal
London Ophthalmic Hospital, Moorfields; Ophthalmic
Surgeon to the National Hospital for the Paralysed
and Epileptic; 54, Queen Anne street, Cavendish
square.

1890 GUTHRIE, LEONARD GEORGE, M.D., B.Ch., Physician to
the Regent's Park Hospital for Epilepsy and Paralysis;
Assistant Physician to the North-West London Hospital; Assistant Physician to the Children's Hospital,
Paddington Green; 15, Upper Berkeley street, Portman square.

Elected

1886   HABERSHON, SAMUEL HERBERT, M.D., Assistant Physician to the Hospital for Consumption, Brompton; 70, Brook street, Grosvenor square.

1885   HAIG, ALEXANDER, M.D., Physician to the Metropolitan Hospital, and to the Royal Hospital for Children and Women; 7, Brook street, Grosvenor square. *Trans.* 6.

1890   HALE, CHARLES DOUGLAS BOWDICH, M.D., 3, Sussex place, Hyde Park.

1881   HALL, FRANCIS DE HAVILLAND, M.D., Physician to the Westminster Hospital; Physician to St. Mark's Hospital; 47, Wimpole street, Cavendish square. *Referee*, 1893-7.

1891   HAMER, WILLIAM HEATON, M.D., Ardeevin, 73, Dartmouth Park Hill, Highgate.

1889   HANDFIELD-JONES, MONTAGU, M.D., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Mary's Hospital; Physician to the British Lying-in Hospital; 35, Cavendish square.

1864   **Harley,** JOHN, M.D., F.L.S., Hon. Physician to St. Thomas's Hospital; Consulting Physician to the London Fever Hospital; 9, Stratford place, Oxford street. S. 1875-7. C. 1879-80. V.P. 1895-7. *Referee*, 1871-4, 1882-95. *Sci. Com.* 1879. *Trans.* 10.

1893   HARLEY, VAUGHAN, M.D., 25, Harley street, Cavendish square.

1892   HAROLD, JOHN, 91, Harley street, Cavendish square.

1880   HARRIS, VINCENT DORMER, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 22, Queen Anne street, Cavendish square. *Referee*, 1899—.

1870   HARRISON, REGINALD, Surgeon to St. Peter's Hospital; 6, Lower Berkeley street, Portman square. C. 1894-5. V.-P. 1898—. *Trans.* 3.

Elected

1870 HAWARD, J. WARRINGTON, *Hon. Treasurer;* Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; 57, Green street, Grosvenor Square. C. 1885. S. 1888-91. V.P. 1894-5. T. (June) 1895—. *Lib. Com.* 1881-4. *Sci. Com.* 1889-91. *Bldg. Com.* (*Sec.*) 1889-92. *Ho. Com.* 1892—. *Trans.* 3.

1891 HAWKINS, HERBERT PENNELL, M.D., B.Ch., Physician to St. Thomas's Hospital; 56, Portland place.

1875 HAYES, THOMAS CRAWFORD, M.D., Physician-Accoucheur and Physician for Diseases of Women and Children to King's College Hospital, and Professor of Midwifery in King's College; Physician for Diseases of Women to the Royal Free Hospital; 17, Clarges street, Piccadilly.

1891 HAYWARD, JOHN ARTHUR, M.D., 17, Lingfield road, Wimbledon. *Pro.* 1.

1865 **Heath,** CHRISTOPHER, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. C. 1880. V.P. 1889. *Lib. Com.* 1870-3. *Trans.* 3.

1895 HENDERSON, EDWARD ERSKINE, B.A., M.B., B.C. [care of W. S. Henderson, Esq., 31, Lombard street, E.C.]; Pembroke lodge, Pembroke gardens, Kensington.

1882 HENSLEY, PHILIP JOHN, M.D., Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; 4, Henrietta street, Cavendish square. *Referee,* 1897—.

1877 HERMAN, GEORGE ERNEST, M.B., Obstetric Physician to, and Lecturer on Midwifery at, the London Hospital; 20, Harley street, Cavendish square. *Referee,* 1892—. *Lib. Com.* 1898—. *Trans.* 1.

1877 HERON, GEORGE ALLAN, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 57, Harley street, Cavendish square.

1891 HERRING, HERBERT T., M.B., B.S., 50, Harley street, Cavendish square.

Elected

1883 HERRINGHAM, WILMOT PARKER, M.D., Assistant Physician,
St. Bartholomew's Hospital; 13, Upper Wimpole
street, Cavendish square. *Trans.* 2.

1893 HERSCHELL, GEORGE, M.D., 76, Wimpole street, Caven-
dish square.

1887 HEWITT, FREDERIC WILLIAM, M.D., Anæsthetist to, and
Instructor in Anæsthetics at, the London Hospital;
Anæsthetist at the Dental Hospital of London; 14,
Queen Anne street, Cavendish square. *Trans.* 3.

1873 HIGGENS, CHARLES, Ophthalmic Surgeon to, and Lecturer
on Ophthalmic Surgery at, Guy's Hospital; 52, Brook
street, Grosvenor square. C. 1894-5. *Trans.* 2.

1890 HILL, G. WILLIAM, M.D., B.Sc., 26, Weymouth street,
Portland place.

1899 HILLIER, ALFRED P., M.D., 30, Wimpole street.

1856 **Holmes,** TIMOTHY, M.A., Consulting Surgeon to St.
George's Hospital; Corresponding Member of the
Société de Chirurgie, Paris; 6, Sussex place, Hyde
Park. C. 1869-70. L. 1873-7. S. 1878-80. V.P.
1881-2. T. 1885-7. P. 1890-92. *Bldg.Com.* (*Chair-
man*) 1889-92. *Referee,* 1866-8, 1872, 1883-4.
Sci. Com. 1867. *Lib. Com.* 1863-5, 1892-5. *Ho. Com.*
1892-8. *Trans.* 8.

1878 HOOD, DONALD WILLIAM CHARLES, M.D., Senior Physician
to the West London Hospital; Examining Physician
for Queen's Messengers, Foreign Office; 43, Green
street, Park lane.

1898 HORDER, T. JEEVES, 29, Constantine road, Hampstead.

1883 HORSLEY, VICTOR ALEXANDER HADEN, F.R.S., Surgeon
to University College Hospital, Surgeon to the
National Hospital for the Paralysed and Epileptic;
25, Cavendish square. *Referee,* 1897—. *Trans.* 1.

Elected

1896 HORTON-SMITH, PERCIVAL, M.D., 15, Upper Brook street. *Sci. Com.* 1897—. *Trans.* 1.

1892 HOWARD, R. J. BLISS, M.D., 31, Queen Anne street, Cavendish square.

1874 HOWSE, HENRY GREENWAY, M.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; 59, Brook street, Grosvenor square. C. 1890. V.P. 1899—. *Sci. Com.* 1879. *Referee*, 1887-89. *Trans.* 3.

1898 HULKE, S. BACKHOUSE, 162, Holland road, Kensington.

1889 HUNTER, WILLIAM, M.D., Senior Assistant Physician to the London Fever Hospital; Curator and Pathologist, Charing Cross Hospital; 103, Harley street.

1873 HUNTER, SIR W. GUYER, M.D., K.C.M.G., Hon. Surgeon to H.M. the Queen; Surgeon-General (Retired) Bombay Army; Consulting Physician to Charing Cross Hospital.

1856 **Hutchinson**, JONATHAN, F.R.S., Consulting Surgeon to, and Emeritus Professor of Surgery at, the London Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Senior Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. P. 1894-5. *Referee*, 1876-81, 1883-94. *Lib. Com.* 1864-5. *Trans.* 14. *Pro.* 2.

1888 HUTCHINSON, JONATHAN, Jun., Surgeon to the London Hospital; 1, Park crescent. *Trans.* 3.

1897 HUTCHISON, R., M.D., Toynbee Hall, Whitechapel.

1871 JACKSON, J. HUGHLINGS, M.D., LL.D., F.R.S., Consulting Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square. C. 1889.

Elected

1883 JACOBSON, WALTER HAMILTON ACLAND, M.Ch.Oxon., Assistant Surgeon to Guy's Hospital; Surgeon to the Royal Hospital for Children and Women; 66, Great Cumberland place, Hyde Park. *Referee*, 1895—. *Lib. Com.* 1896—. *Trans.* 2.

1892 JAMES, EDWIN MATTHEWS, Belgrave Mansions, Grosvenor gardens, and Pavilion, Melrose, N.B.

1897 JAMISON, ARTHUR ANDREW, M.D., 18, Lowndes street, Belgrave square.

1897 JENNER, LOUIS, M.B., 4A, Bloomsbury square.

1883 JESSOP, WALTER H. H., M.B., Ophthalmic Surgeon to St. Bartholomew's Hospital; 73, Harley street.

1881 JOHNSON, GEORGE LINDSAY, M.D., Cortina, Netherhall gardens, South Hampstead, and 36, Finsbury pavement.

1889 JOHNSON, RAYMOND, M.B., B.S., Assistant Surgeon to University College Hospital; Surgeon to the Victoria Hospital for Children; 11, Wimpole street, Cavendish square. *Trans.* 1.

1884 JOHNSTON, JAMES, M.D., 53, Prince's square, Bayswater.

1887 JONES, HENRY LEWIS, M.D., Medical Officer in charge of Electrical Department at St. Bartholomew's Hospital; 61, Wimpole street, Cavendish square.

1896 JONES, L. VERNON, B.A., M.D., B.Ch., 7, Arlington street, St. James's.

1881 JULER, HENRY EDWARD, Ophthalmic Surgeon to St. Mary's Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Consulting Ophthalmic Surgeon to the London Lock Hospital; 23, Cavendish square.

1898 KEEP, A. CORRIE, M.D., C.M., 14, Gloucester place, Portman square.

1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; 56, Grosvenor street, Grosvenor square.

Elected

1898 KELLOCK, THOMAS HERBERT, M.D., B.C., Assistant Surgeon to Middlesex Hospital and to the Hospital for Sick Children; 8, Queen Anne street, Cavendish square.

1884 KESER, JEAN SAMUEL, M.D., Physician to the French Hospital; 11, Harley street, Cavendish square.

1857 **Kiallmark**, HENRY WALTER, 5, Pembridge gardens. C. 1890-91.

1881 KIDD, PERCY, M.D., Physician to the Hospital for Consumption, Brompton; Physician to the London Hospital; 60, Brook street, Grosvenor square. *Trans.* 4.

1851 **Kingdon**, JOHN ABERNETHY, Consulting Surgeon to the Bank of England, Threadneedle street. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.

1896 LANE, JAMES ERNEST, Surgeon to Out-patients, St. Mary's Hospital; 46, Queen Anne street, Cavendish square.

1884 LANE, WILLIAM ARBUTHNOT, M.S., Surgeon to Guy's Hospital and to the Hospital for Sick Children; 21, Cavendish square. *Trans.* 4.

1882 LANG, WILLIAM, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 22, Cavendish square.

1894 LANGDON-DOWN, REGINALD LANGDON, M.B., B.C., 81, Harley street.

1865 **Langton**, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 62, Harley street, Cavendish square. C. 1881-2. V.P. 1895-7. *Referee*, 1885-95. *Lib. Com.* 1879-80, 1888-95. *Trans.* 2.

1898 LATHAM, A. C., M.D., 27, Grosvenor street, Grosvenor square.

Elected

1890   LAW, EDWARD, M.D., C.M., 35, Harley street, Cavendish square.

1898   LAWFORD, J. B., Ophthalmic Surgeon and Lecturer on Ophthalmology, St. Thomas's Hospital; Surgeon to Royal London Ophthalmic Hospital; 99, Harley street.

1888   LAWRENCE, LAURIE ASHER, 4, Queen Anne street.

1893   LAWSON, ARNOLD, Ophthalmic Surgeon to the Children's Hospital, Paddington Green; 12, Harley street, Cavendish square.

1884   LAWSON, GEORGE, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Surgeon to the Royal London Ophthalmic Hospital; Consulting Surgeon to the Middlesex Hospital; 12, Harley street, Cavendish square.

1892   LAZARUS-BARLOW, WALTER SYDNEY, M.D., 55, Penn road villas, West Holloway. *Sci. Com.* 1892—. (Travelling.)

1896   LEE, WILLIAM EDWARD, M.B., Santhapuram, Muswell hill road, Highgate.

1895   LEES, DAVID BRIDGE, M.D., Physician to, and Lecturer on Medicine at, St. Mary's Hospital, and Physician to the Hospital for Sick Children; 22, Weymouth street, Portland place. *Trans.* 2.

1895   LESLIE, ROBERT MURRAY, M.B., Assistant Physician to Royal Hospital for Diseases of the Chest; 53, Queen Anne street.

1897   LEVY, ALFRED G., M.D., 16, York place, Portman square, W.

1886   LEWERS, ARTHUR HAMILTON NICHOLSON, M.D., Obstetric Physician to the London Hospital; 72, Harley street, Cavendish square. *Trans.* 1.

1896   LEWIS, FREDERICK HENRY, M.B., 46, Weymouth street.

1878   LISTER, LORD, D.C.L., LL.D., P.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery in King's College, London; and Consulting Surgeon to King's College Hospital; 12, Park crescent, Regent's Park. C. 1892.

Elected

1891　LITTLE, ERNEST MUIRHEAD, Surgeon to the Natioɪ
Orthopædic Hospital; 40, Seymour street, Portm
square.

1889　LITTLE, JOHN FLETCHER, M.B., 32, Harley street, Cave
dish square.

1881　LOCKWOOD, CHARLES BARRETT, Surgeon to the Gre
Northern Central Hospital; Assistant Surgeon to, aɪ
Lecturer on Surgical and Descriptive Anatomy at, S
Bartholomew's Hospital; 19, Upper Berkeley stree
Portman square. *Trans.* 4.

1897　LOW, HAROLD, 10, Evelyn gardens.

1881　LUCAS, RICHARD CLEMENT, B.S., M.B., Surgeon to, an
Lecturer on Anatomy at, Guy's Hospital; Consultinɡ
Surgeon to the Evelina Hospital for Sick Children
50, Wimpole street, Cavendish square. *Trans.* 3.

1888　LUFF, ARTHUR PEARSON, M.D., B.Sc., Physician to Out-
patients and Lecturer on Medical Jurisprudence aɪ
St. Mary's Hospital; 31, Weymouth street, Portland
place. *Trans.* 1.

1887　LUSH, PERCY J. F., M.B., 4, Maresfield gardens, Hamp-
stead.

1898　LYSTER, C. R. C., Bolingbroke Hospital, Wandsworth
common.

1873　**MacCarthy**, JEREMIAH, M.A., Surgeon to the London
Hospital, late Lecturer on Surgery at the London
Hospital Medical College; 1, Cambridge place, Victoria
road, Kensington. C. 1886-7. *Lib. Com.* 1882-5.
Referee, 1890—.

1867　MAC CORMAC, SIR WILLIAM, Bart., K.C.V.O., M.Ch., D.Sc.,
Surgeon in Ordinary to H.R.H. the Prince of Wales;
Consulting Surgeon and Emeritus Lecturer on Clinical
Surgery to St. Thomas's Hospital; 13, Harley street.
C. 1884-5. V.P. 1896-7. *Referee,* 1889-96. *Lib.
Com* 1895. *Trans.* 1.

1894　MACFADYEN, ALLAN, M.D., B.S., Jenner Institute of Pre-
ventive Medicine, Chelsea bridge.

Elected

1898   McFADYEAN, JOHN, The Royal Veterinary College, Camden Town.

1896   MacGREGOR, ALEXANDER, M.D., 8, Queen street, Mayfair.

1880   McHARDY, MALCOLM MACDONALD, Ophthalmic Surgeon to King's College Hospital, and Professor of Ophthalmic Surgery in King's College, London ; Senior Surgeon to the Royal Eye Hospital, Southwark ; 5, Savile row.

1873   MacKELLAR, ALEXANDER OBERLIN, M.Ch., Surgeon to St. Thomas's Hospital ; Surgeon-in-Chief to the Metropolitan Police Force ; 79, Wimpole street, Cavendish square.

1881   MACKENZIE, STEPHEN, M.D., Physician to the London Hospital ; Physician to the Royal London Ophthalmic Hospital ; 18, Cavendish square. C. 1899—. *Referee*, 1890-9. *Trans.* 1.

1879   MACLAGAN, THOMAS JOHN, M.D., Physician-in-Ordinary to their R.H. the Prince and Princess Christian of Schleswig-Holstein ; 9, Cadogan place, Belgrave square.

1889   MacLEHOSE, NORMAN MacMILLAN, M.B., C.M., Assistant Surgeon, Central London Ophthalmic Hospital ; 13, Queen Anne street, Cavendish square.

1876   MACNAMARA, N. CHARLES, Consulting Surgeon to the Westminster Hospital, and to the Royal Westminster Ophthalmic Hospital ; 13, Grosvenor street. C. 1891-2. *Referee*, 1884-90, 1895-7. *Lib. Com.* 1886-90.

1881   MACREADY, JONATHAN FORSTER CHRISTIAN HORACE, Surgeon to the Great Northern Hospital ; 42, Devonshire street.

1880   MADDICK, EDMUND DISTIN, 5, Cavendish square.

1886   MAGUIRE, ROBERT, M.D., Physician to Out-patients and Joint Lecturer on Pathology at St. Mary's Hospital ; Physician to the Hospital for Consumption, Brompton ; 4, Seymour street, Portman square. *Sci. Com.* 1889—.

Elected

1880 MAKINS, GEORGE HENRY, Surgeon to St. Thomas's Hospital; Consulting Surgeon to the Evelina Hospital for Children; 47, Charles street, Berkeley square. C. 1899—. *Referee*, 1898-9. *Trans.* 2.

1885 MALCOLM, JOHN DAVID, M.B., C.M., Surgeon to the Samaritan Free Hospital; 13, Portman street, Portman square. *Trans.* 2.

1890 MANSON, PATRICK, M.D., C.M., LL.D., Physician to the Seamen's Hospital, Albert Docks; Lecturer on Tropical Medicine at St. George's Hospital; 21, Queen Anne street, Cavendish square.

1855 **Marcet**, WILLIAM, M.D., F.R.S., Flowermead, Wimbledon Park. C. 1871. V.P. 1897-9. *Referee*, 1866-70, 1883-6. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.

1867 MARSH, F. HOWARD, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; 30, Bruton street, Berkeley square. C. 1882-3, 1889. S. 1885-7. V.P. 1891-3. *Lib. Com.* 1880-1. *Trans.* 4.

1891 MARTIN, HENRY CHARRINGTON, M.D., 27, Oxford square.

1884 MARTIN, SIDNEY HARRIS COX, M.D., F.R.S., Assistant Physician to University College Hospital, and to the Hospital for Consumption, Brompton; Professor of Pathology, University College, London; 10, Mansfield street, Portland place.

1892 MASTERS, JOHN ALFRED, M.D., 57, Lexham gardens, Kensington.

1891 MAY, WILLIAM PAGE, M.D., B.Sc., 49, Welbeck street.

891 MERCIER, CHARLES ARTHUR, M.B., Lecturer on Neurology and Insanity at Westminster Hospital; 8, New Court, Lincoln's Inn, and Flower House, Southend, Catford.

..., William ... , M.B., C.M., Surgeon to
... New Hospital for Women and Children :
... square. ... Ho. Com.
...

..., ... , M.D. Surgeon to the German Hos-
... Finsbury square. Trans. ...

... , L.S. ... ... avenue. Hampstead.

... , L.S. ... Walton street. Chelsea.

..., Thomas, M.D. ... , Lecturer. assistant
... ... Lecturer at St. Bartholo-
... Gloucester ..., German square.
...

... Surgeon to the ...
... Hospital for Sick Children,
... Gloucester street. ... ...

... over Jersey

Elected

1888 **Murray,** HUBERT MONTAGUE, M.D., Physician to
patients, and Lecturer on Pathology at, the Ch
Cross Hospital; Physician to the Victoria Hospit
Children; 25, Manchester square.

1898 MURRAY, JOHN, Assistant Surgeon to the Mid
Hospital and to the Paddington Green Chil
Hospital; 110, Harley street.

1880 MURRELL, WILLIAM, M.D., Physician to, and Lectui
Materia Medica, Pharmacology, and Therapeuti
the Westminster Hospital; 17, Welbeck street, C
dish square. *Sci. Com.* 1889—. *Trans.* 1.

1892 MYDDELTON-GAVEY, E. HERBERT, 124, Harley i
Cavendish square, and 16, Broadwater Down,
bridge Wells.

1863 **Myers,** ARTHUR BOWEN RICHARDS, late Brigade-Su:
Brigade of Guards; 43, Gloucester street, Wa
square. C. 1878-9. *Lib. Com.* 1877.

1877 NETTLESHIP, EDWARD, Consulting Ophthalmic Surg
St. Thomas's Hospital; Consulting Surgeon i
Royal London Ophthalmic Hospital; 5, Wi
street, Cavendish square. *Referee,* 1892—.

1864 NUNN, THOMAS WILLIAM, Consulting Surgeon t
Middlesex Hospital; 8, Stratford place, Oxford s

1880 OGILVIE, GEORGE, M.B., B.Sc., Physician to the
pital for Epilepsy and Paralysis, Regent's Parl
Welbeck street, Cavendish square. *Trans.* 1.

1880 OGILVIE, LESLIE, M.B., B.Sc., Physician to the Paddi
Green Children's Hospital; 46, Welbeck street, (
dish square.

1891 OGLE, CYRIL, M.A., M.B., Assistant Physician i
George's Hospital; 96, Gloucester place, Po
square.

1858 OGLE, JOHN WILLIAM, M.D., Consulting Phy
to St. George's Hospital; 96, Gloucester
Portman square. C. 1873. V.P. 1886. R
1864-72. *Trans.* 4.

Elected

1880   MEREDITH, WILLIAM APPLETON, M.B., C.M., Surgeon to
       the Samaritan Free Hospital for Women and Children;
       21, Manchester square.   C. 1897-9.   *Ho. Com.*
       1898-9.   *Trans.* 1.

1894   MICHELS, ERNST, M.D., Surgeon to the German Hos-
       pital; 48, Finsbury square.   *Trans.* 2.

1893   MILEY, MILES, M.B., 21, Belsize avenue, Hampstead.

1891   MOLINE, PAUL, M.B., 42, Walton street, Chelsea.

1873   MOORE, NORMAN, M.D., *Hon. Librarian,* Assistant
       Physician and Lecturer on Medicine to St. Bartholo-
       mew's Hospital; 94, Gloucester place, Portman square.
       C. 1891-2.   L. 1899—.   S. 1896-9.   *Referee,* 1886-90.
       Sci. Com. 1889—.

1878   MORGAN, JOHN HAMMOND, M.A., Surgeon to the Charing
       Cross Hospital, and to the Hospital for Sick Children,
       Great Ormond street; 68, Grosvenor street.   C. 1895-7.
       Dis. Com. 1896-7.   *Trans.* 2.

1894   MORISON, ALEXANDER, M.D., 14, Upper Berkeley
       street.

1874   MORRIS, HENRY, M.A., Surgeon to the Middlesex Hos-
       pital; 8, Cavendish square.   C. 1888-9.   *Referee,*
       1882-7.   *Lib. Com.* 1895-6.   *Trans.* 10.

1879   MORRIS, MALCOLM ALEXANDER, Surgeon to the Skin
       Department of, and Lecturer on Dermatology at, St.
       Mary's Hospital; 8, Harley street, Cavendish square.
       Sci. Com. 1889—.   *Trans.* 1.

1898   MORRISON, JAMES, M.D., St. Bartholomew's Hospital.

1885   MOTT, FREDERICK WALKER, M.D., F.R.S., Assistant
       Physician, Charing Cross Hospital; Pathologist to the
       London County Council; 25, Nottingham place.   *Sci.
       Com.* 1899—.

1899   MUNDY, HERBERT, St. Bartholomew's Hospital.

1896   MURPHY, JAMES KEOGH, M.A., M.B., B.C., 35, Princes
       square, Bayswater.

Elected

1888   **Murray**, HUBERT MONTAGUE, M.D., Physician to Out-patients, and Lecturer on Pathology at, the Charing Cross Hospital; Physician to the Victoria Hospital for Children; 25, Manchester square.

1898   MURRAY, JOHN, Assistant Surgeon to the Middlesex Hospital and to the Paddington Green Children's Hospital; 110, Harley street.

1880   MURRELL, WILLIAM, M.D., Physician to, and Lecturer on Materia Medica, Pharmacology, and Therapeutics at, the Westminster Hospital; 17, Welbeck street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 1.

1892   MYDDELTON-GAVEY, E. HERBERT, 124, Harley street, Cavendish square, and 16, Broadwater Down, Tunbridge Wells.

1863   **Myers**, ARTHUR BOWEN RICHARDS, late Brigade-Surgeon, Brigade of Guards; 43, Gloucester street, Warwick square. C. 1878-9. *Lib. Com.* 1877.

1877   NETTLESHIP, EDWARD, Consulting Ophthalmic Surgeon to St. Thomas's Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital; 5, Wimpole street, Cavendish square. *Referee,* 1892—.

1864   NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1880   OGILVIE, GEORGE, M.B., B.Sc., Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 22, Welbeck street, Cavendish square. *Trans.* 1.

1880   OGILVIE, LESLIE, M.B., B.Sc., Physician to the Paddington Green Children's Hospital; 46, Welbeck street, Cavendish square.

1891   OGLE, CYRIL, M.A., M.B., Assistant Physician to St. George's Hospital; 96, Gloucester place, Portman square.

1858   OGLE, JOHN WILLIAM, M.D., Consulting Physician to St. George's Hospital; 96, Gloucester place, Portman square. C. 1873. V.P. 1886. *Referee,* 1864-72. *Trans.* 4.

Elected

1860 OGLE, WILLIAM, M.D., late Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. V.P. 1887. *Lib. Com.* 1871-5. *Trans.* 5.

1896 OLIVER, GEORGE, M.D., 77, Wimpole street, Cavendish square, and Harrogate.

1892 OPENSHAW, T. HORROCKS, M.B., M.S., Surgeon to, and Lecturer on Anatomy at, the London Hospital; 16, Wimpole street, Cavendish square.

1873 ORD, WILLIAM MILLER, M.D., Consulting Physician to St. Thomas's Hospital; 37, Upper Brook street, Grosvenor square. C. 1889-90. *Sci. Com.* 1889—. *Referee*, 1884-8. *Trans.* 6.

1877 ORMEROD, JOSEPH ARDERNE, M.D., Assistant Physician to St. Bartholomew's Hospital; Physician to the National Hospital for the Paralysed and Epileptic, Queen square; 25, Upper Wimpole street. C. 1897. *Lib. Com.* 1896—. *Trans.* 1.

1875 OSBORN, SAMUEL, 1A, Devonshire street, Portland place, and Maisonnette, Datchet, Bucks.

1879 OWEN, EDMUND, M.B., Senior Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital; Senior Surgeon to the Hospital for Sick Children, Great Ormond street · 64, Great Cumberland place, Hyde park. C. 1896-7. *Trans.* 4.

1882 OWEN, ISAMBARD, M.D., Deputy-Chancellor of the University of Wales; Physician to, and Lecturer on Forensic Medicine at, St. George's Hospital; 40, Curzon street, Mayfair. *Bldg. Com.* 1889-92. *Referee*, 1893, 1895—.

1892 PAGE, H. MARMADUKE, 26, Ashley gardens, Victoria street.

1874 PAGE, HERBERT WILLIAM, M.A., M.C., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 146, Harley street, Cavendish square. C. 1890-91. *Referee*, 1884-89. *Lib. Com.* 1886-8. *Trans.* 4.

Elected

1840 **Paget,** SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Foreign Associate of the Académie de Médecine, Paris; 5, Park square West, Regent's park. C. 1848-9. V.P. 1861. T. 1867. P. 1875-6. *Referee,* 1844-6, 1848, 1851-60, 1862-6, 1868-74. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 12.

1886 PAGET, STEPHEN, Surgeon to the West London Hospital; Surgeon to the Throat and Ear Department of the Middlesex Hospital; 70, Harley street.

1895 PARKER, CHARLES ARTHUR, 41, Queen Anne street, Cavendish square.

1873 PARKER, ROBERT WILLIAM, Senior Surgeon to the East London Hospital for Children; Senior Surgeon to the German Hospital; 13, Welbeck street, Cavendish square. C. 1888-9, 1899—. S. 1895-8. *Bldg. Com.* 1889-92. *Referee,* 1891-5. *Lib. Com.* 1885-87, 1892-5, 1898-9. *Ho. Com.* 1892-5, 1899—. *Trans.* 4.

1889 PARSONS, J. INGLIS, M.D., Physician to the Chelsea Hospital for Women; 3, Queen street, Mayfair.

1883 PASTEUR, WILLIAM, M.D., Senior Assistant Physician to the Middlesex Hospital; Consulting Physician to the North-Eastern Hospital for Children; 4, Chandos street, Cavendish square.

1891 PATERSON, WILLIAM BROMFIELD, 7A, Manchester square.

1891 PATON, EDWARD PERCY, M.D., M.S., 84, Park street, Grosvenor square.

1865 **Pavy,** FREDERICK WILLIAM, M.D., LL.D., F.R.S., Consulting Physician to Guy's Hospital; 35, Grosvenor street. C. 1883-4. V.P. 1893-4. *Referee,* 1871-82. *Trans.* 1.

1869 PAYNE, JOSEPH FRANK, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. C. 1887. *Referee,* 1890—. *Sci. Com.* 1879. *Lib. Com.* 1878-85, 1889—.

Elected

1894   PEGLER, L. HEMINGTON, M.D., 27, Welbeck street.

1898   PENDLEBURY, HERBERT STRINGFELLOW, M.B., B.C., St.
George's Hospital (Highfield House, Standish, Wigan,
Lancs.).

1887   PENROSE, FRANCIS GEORGE, M.D., Physician to St.
George's Hospital and to the Hospital for Sick
Children, Great Ormond street; 84, Wimpole street,
Cavendish square. *Sci. Com.* 1889—.

1897   PERRAM, CHARLES HERBERT, M.D., 22, Manchester
square.

1890   PERRY, EDWIN COOPER, M.D., Physician to, and Demon-
strator of Pathology at, Guy's Hospital; The Super-
intendent's House, Guy's Hospital.

1895   PHEAR, ARTHUR G., M.D., Assistant Physician and Patho-
logist to the Metropolitan Hospital; 47, Weymouth
street, Portland place. *Trans.* 1.

1883   PHILLIPS, CHARLES DOUGLAS F., M.D., LL.D., 10, Hen-
rietta street, Cavendish square.

1884   PHILLIPS, GEORGE RICHARD TURNER, J.P., 28, Palace
Court, Bayswater hill.

1888   PHILLIPS, JOHN, M.A., M.D., Obstetric Physician, King's
College Hospital; Lecturer on Practical Obstetrics in
King's College; Physician to the British Lying-in
Hospital; 68, Brook street, Grosvenor square.
Trans. 1.

1898   PHILLIPS, L. C. POWELL, St. Bartholomew's Hospital.

1889   PHILLIPS, SIDNEY, M.D., Physician and Lecturer on
Medicine at St. Mary's Hospital; Senior Physician to
the London Fever Hospital, and to the Lock Hospital;
62, Upper Berkeley street, Portman square. *Trans.* 1.

1867   PICK, THOMAS PICKERING, Consulting Surgeon to St.
George's Hospital; 18, Portman street, Portman
square. C. 1884-5. V.P. 1893-4. *Referee,* 1882-3,
1891-93. *Sci. Com.* 1870, 1889—. *Lib. Com.*
1879-81.

Elected

1884 PITT, GEORGE NEWTON, M.D., Physician to, and Pathologist at, Guy's Hospital; 15, Portland place. *Trans.* 1. *Referee*, 1897—.

1889 PITTS, BERNARD, M.A., M.C., Surgeon to St. Thomas's Hospital and Lecturer on Surgery; Surgeon to the Hospital for Sick Children, Great Ormond street; 109, Harley street, Cavendish square. *Referee,* 1897—.

1885 POLAND, JOHN, Surgeon to the City Orthopædic Hospital and Miller Hospital, Greenwich; 4, St. Thomas's street, London Bridge.

1884 POLLARD, BILTON, B.S., Surgeon to University College Hospital; Consulting Surgeon to the North-Eastern Hospital for Children; 24, Harley street, Cavendish square. *Trans.* 1.

1865 **Pollock, JAMES EDWARD,** M.D., Consulting Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. C. 1882-3. V.P. 1896-7. *Referee*, 1872-81.

1894 POLLOCK, WILLIAM RIVERS, M.B., B.C., Assistant Obstetric Physician to the Westminster Hospital; 56, Park street, Grosvenor square.

1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence and Clinical Medicine in University College, London; Physician to University College Hospital; 32, Wimpole street, Cavendish square. C. 1890-91. *Referee*, 1887-9, 1892—. *Lib. Com.* 1895—. *Trans.* 2.

1887 POWER, D'ARCY, M.A., M.B., Assistant Surgeon at St. Bartholomew's Hospital; Surgeon to the Victoria Hospital for Children, Chelsea; 10A, Chandos street, Cavendish Square. *Lib. Com.* 1896—. *Trans.* 2.

Elected

1867   **Power**, HENRY, Consulting Ophthalmic Surgeon to St. Bartholomew's Hospital ; 37A, Great Cumberland place, Hyde Park. C. 1882-3. V.P. 1892-3. *Referee*, 1870-81, 1891-2. *Sci. Com.* 1870, 1889—. *Lib. Com.* 1872-8.

1857   **Priestley**, SIR WILLIAM OVEREND, M.D., LL.D., M.P., Consulting Physician to King's College Hospital, and to the West London Hospital and the British Lying-in Hospital ; 17, Hertford street, Mayfair. C. 1874-5. V.P. 1884-5. *Referee*, 1867-73, 1877-83. *Sci. Com.* 1863.

1883   PRINGLE, JOHN JAMES, M.B., C.M., Physician in Charge of Skin Department at the Middlesex Hospital; 23, Lower Seymour street, Portman square. *Trans.* 2.

1874   PURVES, WILLIAM LAIDLAW, Aural Surgeon to Guy's Hospital ; 20, Stratford place, Oxford street. *Trans.* 2.

1877   PYE-SMITH, PHILIP HENRY, M.D., F.R.S.. Physician to, and Lecturer on Medicine at, Guy's Hospital; 48, Brook street, Grosvenor square. C. 1893-4. *Lib. Com.* 1887-93, 1899—. *Referee*, 1897—. *Trans.* 1.

1898   RAMSAY, HERBERT MURRAY, 35A, Hertford street.

1893   RANKIN, GUTHRIE, 4, Chesham street. Belgrave square.

1899   RAWLING, LOUIS BATHE, M.B., B.C., St. Bartholomew's Hospital.

1892   RAYNER, HENRY, M.D., Lecturer on Psychological Medicine to St. Thomas's Hospital ; 16, Queen Anne street, Cavendish square.

1869   READ, THOMAS LAURENCE, 11, Petersham terrace, Queen's gate.

1891   REECE, RICHARD JAMES, 62, Addison gardens.

1891   RENDEL, ARTHUR BOWEN, M.A., M.B., B.C., 43, Albion street, Hyde Park.

Elected

1887 RICHARDSON, GILBERT, M.A., M.D., Hillside, Putney h

1863 RINGER, SYDNEY, M.D., F.R.S., Holme Professor
      Clinical Medicine in University College, Lond(
      and Physician to University College Hospital; 1
      Cavendish place, Cavendish square.   C. 1881·
      Referee, 1873-80, 1889-97.   *Trans.* 6.

1896 ROBERTS, CHARLES HUBERT, M.D., 21, Welbeck street.

1893 ROBERTS, D. WATKIN, M.D., 56, Manchester stree
      Manchester square.

1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Med.
      cine, and of Clinical Medicine, in University Colleg(
      London; Physician to University College Hospital
      Consulting Physician to the Hospital for Consumption
      Brompton; 102, Harley street, Cavendish square
      C. 1894-5.   *Sci. Com.* 1889—.

1898 ROBERTSON, F. W., 54, Bernard street, Russell square.

1896 ROBINSON, HENRY BETHAM, M.S., Assistant Surgeon to,
      and Surgeon in Charge of the Throat Department,
      St. Thomas's Hospital; Assistant Surgeon to the East-
      London Hospital for Children, Shadwell; 1, Upper
      Wimpole street.

1890 ROLLESTON, HUMPHRY DAVY, M.D., Senior Assistant
      Physician to, and Lecturer on Pathology at, St.
      George's Hospital; Senior Physician to Out-patients,
      Victoria Hospital for Children; 112, Harley street,
      Cavendish square.

1857 **Rose**, HENRY COOPER, M.D., 16, Warwick road, Maida
      Vale.   C. 1886-7.   *Trans.* 1.

1883 ROSE, WILLIAM, M.B., Professor of Clinical Surgery in
      King's College; Senior Surgeon to King's College
      Hospital; Consulting Surgeon to the Royal Free Hos-
      pital; 17, Harley street, Cavendish square.

Elected

1892 SCHORSTEIN, GUSTAVE, M.A., M.B., B.Ch., D.P.H., Assistant Physician to the London Hospital, and to the Hospital for Consumption, Brompton; 11, Portland place.

1899 SCOTT, LINDLEY MARCROFT, M.D., 98, Sloane street.

1882 SCRIVEN, JOHN BARCLAY, Brigade Surgeon, Bengal (retired); 95, Oxford gardens, North Kensington.

1863 **Sedgwick**, WILLIAM, 101, Gloucester place, Portman square. C. 1884-5. *Trans.* 3.

1892 SEGUNDO, CHARLES SEMPILL DE, M.B., B.S., 6, Brook street, Hanover square.

1892 SELWYN-HARVEY, JOHN STEPHENSON, M.D., 1, Astwood road, Cromwell road.

1877 SEMON, SIR FELIX, M.D., Physician for Diseases of the Throat to the National Hospital for Epilepsy and Paralysis, Queen square; 39, Wimpole street, Cavendish square. C. 1895-7. *Lib. Com.* 1894-5. *Trans.* 3.

1894 SEWILL, JOSEPH SEFTON, 9A, Cavendish square.

1882 SHARKEY, SEYMOUR JOHN, M.D., Physician to, and Joint Lecturer on Medicine at, St. Thomas's Hospital; 22, Harley street, Cavendish square. C. 1899—. *Referee,* 1897-9. *Trans.* 2.

1886 SHAW, LAURISTON ELGIE, M.D., Physician to Guy's Hospital; 10, St. Thomas's street, Southwark.

1884 SHEILD, ARTHUR MARMADUKE, M.B., B.C., Assistant Surgeon to St. George's Hospital; 4, Cavendish place. *Referee,* 1897—. *Trans.* 6.

1896 SHORE, THOMAS WILLIAM, M.D., Heathfield, Alleyn park, Dulwich.

1893 SIBLEY, WALTER KNOWSLEY, M.D., B.C., Senior Physician to Out-patients, North-West London Hospital; 1, Duke street mansions, Grosvenor square.

Elected

1848   **Sieveking,** Sir Edward Henry, M.D., LL.D., F.S.A. Physician-in-Ordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's and the Lock Hospitals; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. P. 1888-9. *Referee,* 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.

1886   Silcock, Arthur Quarry, B.S., Surgeon in charge of Out-patients, and Teacher of Operative Surgery, St. Mary's Hospital; Surgeon to the Royal London Ophthalmic Hospital; 52, Harley street, Cavendish square. *Lib. Com.* 1895—.

1842   **Simon,** Sir John, K.C.B., F.R.S., Hon. M.D.Dublin, 1887, Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee,* 1851-3, 1866-81. *Trans.* 1.

1892   Sims, Francis Manley Boldero, 12, Hertford street, Mayfair.

1894   Slater, Charles, M.B., 81, St. Ermin's mansions, Westminster.

1896   Sloane, John Stretton, M.B., B.S., B.Sc., 3, Montagu mansions, Portman square.

1890   Smale, Morton, Surgeon Dentist to St. Mary's Hospital; 22A, Cavendish square.

1879   Smith, E. Noble, Surgeon to the City Orthopædic Hospital; Surgeon to All Saints' Children's Hospital; Orthopædic Surgeon to the British Home for Incurables; 24, Queen Anne street, Cavendish square.

1881   Smith, Eustace, M.D., Physician to H.M. the King of the Belgians; Physician to the East London Children's Hospital, and to the Victoria Park Hospital for Diseases of the Chest; 15, Queen Anne street, Cavendish square. C. 1899—.

Elected

1838 **Smith**, HENRY SPENCER, Consulting Surgeon to St.
Mary's Hospital; 92, Oxford terrace, Hyde Park.
C. 1854.   S. 1855-8.   V.P. 1859-60.   T. 1865.
Referee, 1851-3, 1862-4, 1866-78.   *Lib. Com.* 1847.

1866 SMITH, HEYWOOD, M.A.,M.D., 18, Harley street, Cavendish
square.

1889 SMITH, ROBERT PERCY, M.D., B.S., Lecturer on Psycho-
logical Medicine, Charing Cross Hospital; 36, Queen
Anne street.

1892 SMITH, SOLOMON CHARLES, M.D., Four Oaks, Walton-on-
Thames.

1863 **Smith**, SIR THOMAS, Bart., Surgeon Extraordinary to
H.M. the Queen; Consulting Surgeon to St. Bartholo-
mew's Hospital; 5, Stratford place, Oxford street.
S. 1870-2.   C. 1875-6.   V.P. 1887-8.   *Referee*, 1873-4,
1880-6.   *Sci. Com.* 1867.   *Trans.* 4.

1872 SMITH, THOMAS GILBART, M.D., Physician to the London
Hospital; Physician to the Royal Hospital for Diseases
of the Chest, City road; 68, Harley street, Cavendish
square.   C. 1890.   *Trans.* 1.

1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital
Society, Greenwich.

1874 *Smith*, WILLIAM ROBERT, M.D., D.Sc., F.R.S.Edin., Bar-
rister-at-Law, Professor of Forensic Medicine, and
Director of the Laboratories of State Medicine in
King's College, London; Medical Officer to the
School Board for London; 74, Great Russell street.
Trans. 1.

1865 **Southey**, REGINALD, M.D., Commissioner in Lunacy; 32,
Grosvenor road, Westminster.   C. 1881-2.   S. 1883.
Referee, 1873-80.   *Trans.* 1.

1889 SPENCER, HERBERT R., M.D., B.S., Professor of Mid-
wifery in University College; Obstetric Physician to
University College Hospital; 104, Harley street.
Referee, 1894—.

1887 SPENCER, WALTER GEORGE, M.B., M.S., Surgeon to, and
Lecturer on Physiology at, the Westminster Hospital;
35, Brook street, Grosvenor square.   *Trans.* 2.

Elected

1888 SPICER, ROBERT HENRY SCANES, M.D., Surgeon to the Department for Diseases of the Throat, St. Mary's Hospital; 28, Welbeck street, Cavendish square.

1890 SPICER, WILLIAM THOMAS HOLMES, M.B., 47, Welbeck street, Cavendish square.

1875 SPITTA, EDMUND JOHNSON, Ivy House, 31, South Side, Clapham Common, Surrey.

1851 **Spitta,** ROBERT JOHN, M.D., 2, The Sweep, East Side, Clapham Common, Surrey. C. 1878-9. *Trans.* 1.

1885 SQUIRE, JOHN EDWARD, M.D., Physician to the North London Hospital for Consumption; 122, Harley street, Cavendish square. *Trans.* 2.

1897 STAINER, EDWARD, M.A., M.B., 43, Vincent square.

1856 **Stocker,** ALONZO HENRY, M.D., Peckham House, Peckham.

1884 STONHAM, CHARLES, Surgeon to, and Lecturer on Surgery and Teacher of Operative Surgery at, the Westminster Hospital; Surgeon to the Poplar Hospital for Accidents; 4, Harley street, Cavendish square.

1896 SUTHERLAND, GEORGE ALEXANDER, M.D., Physician to Paddington Green Children's Hospital; Assistant Physician to the North-West London Hospital; 9, Old Cavendish street.

1871 **Sutherland,** HENRY, M.D., 21, New Cavendish street.

1883 SUTTON, JOHN BLAND, Assistant Surgeon to the Middlesex Hospital; Surgeon to the Chelsea Hospital for Women; 48, Queen Anne street, Cavendish square. *Trans.* 6.

1896 SWAN, CHARLES ROBERT JOHN ATKIN, M.B., B.Ch., 4, Devonport street, Hyde Park.

1890 SYERS, HENRY WALTER, M.D., 40, Wimpole street.

1886 SYMONDS, CHARTERS JAMES, M.S., M.D., Surgeon to, and Surgeon in charge of the Throat Department at, Guy's Hospital; 58, Portland place.

Elected

1875   TAY, WAREN, Senior Surgeon to the London Hospital, to the Royal London Ophthalmic Hospital, and to the Hospital for Diseases of the Skin, Blackfriars; Consulting Surgeon to the North-Eastern Hospital for Children ; 4, Finsbury square.

1873   TAYLOR, FREDERICK, M.D., *Trustee;* Physician to, and Lecturer on Medicine at, Guy's Hospital ; Consulting Physician to the Evelina Hospital for Sick Children ; 20, Wimpole street, Cavendish square. S. 1889-93. C. 1894-6. *Sci. Com.* 1889—. *Referee,* 1887-8, 1899. *Trans.* 3.

1893   TAYLOR, JAMES, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic ; Physician to the North-Eastern Hospital for Children, and to the National Orthopædic Hospital; 49, Welbeck street, Cavendish square. *Trans.* 1.

1890. TAYLOR, SEYMOUR, M.D., Assistant Physician, West London Hospital ; 16, Seymour street, Portman square.

1859   TEGART, EDWARD, 60, Scarsdale Villas, Kensington. C. 1888-9.

1874   THIN, GEORGE, M.D., 63, Harley street, Cavendish square. C. 1893-4. *Trans.* 14.

1862   Thompson, EDMUND SYMES, M.D., Consulting Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine ; 33, Cavendish square. S. 1871-4. C. 1878-9. *Sci. Com.* 1889—. *Referee,* 1876-7. *Trans.* 1.

1852. Thompson, SIR HENRY, Bart., Surgeon-Extraordinary to H.M. the King of the Belgians ; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital ; 35, Wimpole street, Cavendish square. V.P. 1888. 1869. *Trans.* 8.

Elected

1862   Thompson, REGINALD EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 47, Park street, Grosvenor square. C. 1879. S. 1880-82. V.P. 1883-4. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.

1892   THOMSON, STCLAIR, M.D., Physician to the Throat Hospital, Golden Square; Surgeon to the Royal Ear Hospital, London; 28, Queen Anne street, Cavendish square. *Trans.* 1.

1892   THORNE, WILLIAM BEZLY, M.D., 53, Upper Brook street.

1899   THURSFIELD, JAMES HUGH, M.B., 10, Bentinck street.

1889   TIRARD, NESTOR ISIDORE CHARLES, M.D., Professor of Materia Medica and Therapeutics, King's College; Physician to King's College Hospital, and Physician to the Evelina Hospital for Sick Children; 74, Harley street, Cavendish square.

1872   TOMES, CHARLES SISSMORE, M.A., F.R.S., 9, Park crescent, Portland place. C. 1887. V.P. 1897—. *Lib. Com.* 1879.

1882   TOOTH, HOWARD HENRY, M.D., Physician to the National Hospital for the Paralysed and Epileptic, Queen square; Assistant Physician to St. Bartholomew's Hospital; 34, Harley street, Cavendish square. *Sci. Com.* 1896 —.

1879   TREVES, FREDERICK, Surgeon-in-Ordinary to H.R.H. the Duke of York; Consulting Surgeon to the London Hospital; 6, Wimpole street, Cavendish square. C. 1895-6. *Referee*, 1890-95. *Sci. Com.* 1889-95. *Trans.* 5.

1859   Truman, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1897   TUNNICLIFFE, FRANCIS WHITTAKER, M.D., 6, Devonshire street, Portland place.

1889   TURNBULL, GEORGE LINDSAY, M.D., Grove House, 76, Ladbroke grove.

Elected

1875 TURNER, FRANCIS CHARLEWOOD, M.D., Physician to the London Hospital; Consulting Physician to the North-Eastern Hospital for Children; 15, Finsbury square. C. 1895-7.

1882 TURNER, GEORGE ROBERTSON, Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; Visiting Surgeon to the Seamen's Hospital, Greenwich; 49, Green street, Park lane.

1898 TURNER, WILLIAM, M.B., B.S., Assistant Surgeon, Westminster Hospital; 53, Queen Anne street, Cavendish square.

1896 TURNER, WILLIAM ALDREN, M.D., Assistant Physician West London Hospital; 13, Queen Anne street, Cavendish square.

1896 TURNEY, HORACE GEORGE, M.D., Joint Lecturer on Pathology and Assistant Physician to St. Thomas's Hospital; 68, Portland place. *Trans.* 1.

1892 TWEEDY, JOHN, Professor of Ophthalmic Medicine and Surgery in University College, Ophthalmic Surgeon to University College Hospital, and Surgeon to the Royal London Ophthalmic Hospital; 100, Harley street, Cavendish square.

1876 VENN, ALBERT JOHN, M.D., 63, Grosvenor street.

1870 VENNING, EDGCOMBE, 30, Cadogan place. C. 1898—.

1865 Vernon, BOWATER JOHN, Senior Ophthalmic Surgeon to St. Bartholomew's Hospital; 14, Clarges street, Piccadilly.

867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy, and Senior Physician to the French Hospital and Dispensary, Shaftesbury Avenue; 19A, Hanover square.

1891 VOELCKER, ARTHUR FRANCIS, M.D., B.S., Assistant Physician to, and Lecturer on Pathology at, the Middlesex Hospital; Assistant Physician, Hospital for Sick Children, Great Ormond street; 31, Harley street.

Elected

1896   WAGGETT, ERNEST, M.B., B.C., Assistant Surgeon, London Throat Hospital; 45, Upper Brook street.

1884   WAKLEY, THOMAS, jun., 5, Queen's Gate, South Kensington.

1896   WALDO, FREDERICK JOSEPH, M.D., 1, Plowden Buildings, Temple.

1883   WALLER, AUGUSTUS, M.D., F.R.S., Lecturer on Physiology, St. Mary's Hospital; Weston Lodge, 16, Grove End road, St. John's Wood. *Referee*, 1895—.

1888   WALLIS, FREDERICK CHARLES, M.B., B.C., Assistant Surgeon to the Charing Cross Hospital; 26, Welbeck street, Cavendish square.

1896   WALSHAM, HUGH, M.A., M.D., Assistant Physician to the City of London Hospital for Diseases of the Chest; Assistant Medical Officer in Electrical Department, St. Bartholomew's Hospital; 114, Harley street, Cavendish square.

1873   WALSHAM, WILLIAM JOHNSON, C.M., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Consulting Surgeon to the Metropolitan Hospital; 77, Harley Street, Cavendish square. C. 1888-9. *Referee*, 1895—. *Lib. Com.* 1882-5. *Trans.* 8.

1899   WALTERS, FREDERICK RUFENACHT, M.D., 60, Welbeck street, Cavendish square.

1880   WARD, ALLAN OGIER, M.D., Lansdowne House, High road, Tottenham.

1890   WARD, ARTHUR HENRY, Surgeon to Out-patients, Lock Hospital; 31, Grosvenor street.

1894   WARD-HUMPHREYS, GEORGE HERBERT, 16, Cavendish square.

1891   WARING, H. J., M.B., M.S., B.Sc., Surgical Registrar and Demonstrator of Operative Surgery, St. Bartholomew's Hospital; Surgeon, Metropolitan Hospital; 9, Upper Wimpole street.

Elected

1877 WARNER, FRANCIS, M.D., Physician to, and Lecturer on Materia Medica and Therapeutics at, the London Hospital ; 5, Prince of Wales terrace, Kensington Palace. C. 1899—. *Trans.* 2.

1889 WASHBOURN, JOHN WICHENFORD, M.D., Physician to, Joint Lecturer on Physiology, and Lecturer on Bacteriology at, Guy's Hospital ; Physician to the London Fever Hospital ; 6, Cavendish place. *Trans.* 1.

1894 WATERHOUSE, HERBERT FURNIVALL, C.M., Senior Assistant Surgeon and Lecturer on Anatomy, Charing Cross Hospital ; Surgeon, Victoria Hospital for Children ; 81, Wimpole street.

1861 Watson, WILLIAM SPENCER, M.B., 1, Duke street, Manchester square. C. 1883-4. *Trans.* 1.

1879 DE WATTEVILLE, ARMAND, M.D., 30, Welbeck street, Cavendish square.

1892 WEAVER, FREDERICK POYNTON, M.D., Cedar Lawn, Hampstead heath.

1891 Weber, FREDERIC PARKES, M.D., Physician to the German Hospital, Dalston ; 19, Harley street.

1857 WEBER, SIR HERMANN, M.D., Consulting Physician to the German Hospital ; 10, Grosvenor street, Grosvenor square. C. 1874-5. V.P. 1885-6. *Sci. Com.* 1889—. *Referee*, 1869-73, 1878-84. *Lib. Com.* 1864-73. *Trans.* 6.

1896 WEIR, ARTHUR NESHAM, M.B., 55, St. Charles square, Bayswater.

1895 WELLS, SYDNEY RUSSELL, M.D., 24, Somerset street, Portman square.

1877 WEST, SAMUEL, M.D., Assistant Physician to St. Bartholomew's Hospital ; Senior Physician to the Royal Free Hospital ; 15, Wimpole street, Cavendish square. C. 1894-5. *Lib. Com.* 1892-4. *Trans.* 7.

Elected

1888   WETHERED, FRANK JOSEPH, M.D., Assistant Physician to the Hospital for Consumption, Brompton; 83, Harley street, Cavendish square. *Trans.* 1.

1881   WHARRY, ROBERT, M.D., 6, Gordon square.

1875   WHIPHAM, THOMAS TILLYER, M.D., Consulting Physician to St. George's Hospital; 11, Grosvenor street, Grosvenor square. C. 1892-3.

1891   WHITE, CHARLES PERCIVAL, M.B., B.C., 144, Sloane street.

1881   WHITE, WILLIAM HALE, M.D., Physician to, and Lecturer on Materia Medica at, Guy's Hospital; 65, Harley street, Cavendish square. *Referee,* 1888-97, 1899—. *Trans.* 4.

1890   WHITE-COOPER, W. G. O., M.B., 5, Courtfield road, Gloucester road.

1897   WHITFIELD, ARTHUR, M.D., 12, Upper Berkeley street.

1899   WHITING, ARTHUR J., M.D., 142, Harley street.

1877   WHITMORE, WILLIAM TICKLE, Consulting Surgeon to the Gordon Hospital for Diseases of the Rectum; 7, Arlington street, Piccadilly.

1863   **Wilks,** SIR SAMUEL, Bart., M.D., LL.D., F.R.S., Physician-Extraordinary to H.M. the Queen, Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught; Consulting Physician to Guy's Hospital; 72, Grosvenor street. *Referee,* 1872–81.

1890   WILLCOCKS, FREDERICK, M.D., Physician to Out-Patients, and Lecturer on Materia Medica and Therapeutics, at the Charing Cross Hospital; Physician to the Evelina Hospital for Sick Children; 14, Mandeville place, Manchester square.

1865   **Willett,** ALFRED, *Trustee;* Surgeon to St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. C. 1880-81. V.P. 1890-91. *Referee,* 1882-89, 1892—. *Bldg. Com.* 1889-92. *Ho. Com.* 1892-8. *Trans.* 2.

Elected

1887 WILLETT, EDGAR, M.B., 25, Welbeck street, Cavendish square.

1888 WILLIAMS, CAMPBELL, 18, Queen Anne street.

1866 **Williams**, CHARLES THEODORE, M.A., M.D., *Trustee for Debenture-holders;* Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 2, Upper Brook street, Grosvenor square. C. 1884-5. *Referee,* 1888—. *Lib. Com.* 1880-3. *Sci. Com.* 1889—. *Trans.* 6.

1881 WILLIAMS, DAWSON, M.D., Physician to the East London Hospital for Children ; 101, Harley street. *Trans.* 1.

1872 WILLIAMS, SIR JOHN, Bart., M.D., Physician-Accoucheur to H.R.H. the Duchess of York, Physician to H.R.H. the Princess Beatrice; Emeritus Professor of Obstetric Medicine, University College, London; Consulting Obstetric Physician to University College Hospital; 63, Brook street, Grosvenor square. C. 1891. *Referee,* 1878-90. *Lib. Com.* 1876-82.

1890 WILLS, WILLIAM ALFRED, M.D., Assistant Physician to the Westminster Hospital ; Senior Physician to the North-Eastern Hospital for Children; 29, Lower Seymour street, Portman square.

1879 WOAKES, EDWARD, M.D., Senior Aural Surgeon to the London Hospital; 78, Harley street, Cavendish square.

1887 WOOD, THOMAS OUTTERSON, M.D., Senior Physician to the West End Hospital for Nervous Diseases; 40, Margaret street, Cavendish square.

1891 WOODFORDE, ALFRED POWNALL, 160, Goldhawk road.

1890 WYNTER, WALTER ESSEX, M.D., Assistant Physician to the Middlesex Hospital; 30, Upper Berkeley street, Portman square.

LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

DATE OF ELECTION

1838 Henry Spencer Smith.
1840 Sir James Paget, Bt., F.R.S.
1842 Sir John Simon, K.C.B., F.R.S.
1845 Sir Edwin Saunders.
 Edward U. Berry.
1848 Sir Edward H Sieveking, M.D.
 John Clarke, M.D.
1849 C. H. F. Routh, M.D.
1851 John Birkett.
 John A. Kingdon.
 Bernard E. Brodhurst.
 Robert J. Spitta, M.D.
1852 William Adams.
 Sir Henry Thompson.
1853 Robert Brudenell Carter.
1854 Sir Alfred B. Garrod, M.D., F.R.S.
1855 William Marcet, M.D., F.R.S.
1856 William Bird.
 Jonathan Hutchinson, F.R.S.
 Timothy Holmes.
 Alonzo H. Stocker, M.D.
1857 Sir William Overend Priestley, M.D.
 Sir Hermann Weber, M.D.
 Henry Cooper Rose, M.D.
 Henry Walter Kiallmark.
1858 John William Ogle, M.D.
1859 Wm. Howship Dickinson, M.D.
 Edwin Thomas Truman.
 Richard Barwell.
 Edward Tegart.

1860 William Ogle, M.D.
 Thomas Bryant.
 John Couper.
 Henry Howard Hayward.
1861 William Spencer Watson.
1862 Lionel Smith Beale, M.B., F.R.S.
 Edmund Symes Thompson, M.D.
 Reginald Edward Thompson, M.D.
 George Cowell.
1863 Sir Samuel Wilks, Bt., M.D., F.R.S.
 Samuel Fenwick, M.D.
 Julius Althaus, M.D.
 Sydney Ringer, M.D., F.R.S.
 Sir Thomas Smith, Bart.
 Arthur B. R. Myers.
 William Sedgwick.
1864 John Harley, M.D.
 Thomas William Nunn.
1865 James Edward Pollock, M.D.
 Reginald Southey, M.D.
 George Fielding Blandford, M.D.
 Sir Dyce Duckworth, M.D.
 Frederick W. Pavy, M.D., F.R.S.
 John Langton.
 Frederick James Gant.
 Alfred Willett.
 Bowater John Vernon.
 Alfred Cooper.
 Christopher Heath.
1866 Samuel Jones Gee, M.D.

1866 Charles Theodore Williams, M.D.
Heywood Smith, M.D.
William Selby Church, M.D.
1867 Achille Vintras, M.D.
Sir R. Douglas-Powell, Bart., M.D.
F. Howard Marsh.
Henry Power.
Sir William MacCormac, Bart.
Thomas Pickering Pick.
1868 H. Charlton Bastian, M.D., F.R.S.
Sir W. H. Broadbent, Bart., M.D.
Thomas Buzzard, M.D.
Walter Butler Cheadle, M.D.
T. Henry Green, M.D.
William Chapman Grigg, M.D.
John Croft.
George Eastes.
1869 Joseph Frank Payne, M.D.
Arthur E. Sansom, M.D.
Thomas Laurence Read.
1870 J. Warrington Haward.
Edgcombe Venning.
Clement Godson, M.D.
Reginald Harrison.
Robert Leamon Bowles, M.D.
1871 William Cayley, M.D.
T. Lauder Brunton, M.D., F.R.S.
J. Hughlings Jackson. M.D., F.R.S.
Henry Sutherland, M.D.
George Vivian Poore, M.D.
Benjamin Duke, M.D.
Philip Frank, M.D.
1872 T. Gilbart-Smith, M.D.
George B. Brodie, M.D.
Sir John Williams, Bart., M.D.
Sir J. Fayrer, M.D., F.R.S.
Charles S. Tomes, M.A., F.R.S.
Sir William Bartlett Dalby.
1873 William Miller Ord, M.D.
Frederick Taylor, M.D.
Norman Moore, M.D.
John Curnow, M.D.
Sir William R. Gowers, M.D., F.R.S.
Sir Wm. Guyer Hunter, M.D.
Jeremiah McCarthy.
Wm. Johnson Smith.
Robert William Parker.
Alex. O. MacKellar.
Henry T. Butlin.
Charles Higgens.
William J. Walsham.
1874 Alfred Lewis Galabin, M.D.
George Thin, M.D.
John Mitchell Bruce, M.D.

1874 Henry Morris.
William Laidlaw Purves.
William Harrison Cripps.
Henry G. Howse. M.S.
Herbert William Page.
Frederic Durham.
William Robert Smith, M.D.
1875 Thomas T. Whipham, M.D.
Francis Charlewood Turner, M.]
Thomas Crawford Hayes, M.D.
Charles Henry Carter, M.D.
Waren Tay.
Edmund J. Spitta.
Samuel C. Osborn.
Fletcher Beach, M.B.
1876 Thomas Barlow, M.D.
Wm. Lewis Dudley, M.D.
Albert J. Venn, M.D.
N. Charles Macnamara.
John N. C Davies-Colley, M.C.
1877 Sir Felix Semon, M.D.
Sidney Coupland, M.D.
Francis Warner, M.D.
William Ewart, M.D.
Alfred Pearce Gould, M.S.
Rickman J. Godlee, M.S.
Alban H. G. Doran.
George Ernest Herman, M.B.
Samuel West, M.D.
John Abercrombie, M.D.
George Allan Heron, M.D.
Joseph A. Ormerod, M.D.
P. Henry Pye-Smith, M.D., F.R
Edward Nettleship.
William Henry Bennett.
William T. Whitmore.
1878 Sir Jas. Crichton-Browne, M.D.
Fred. T. Roberts, M.D.
Lord Lister, F.R.S.
Clinton T. Dent.
John H. Morgan.
Donald W. Charles Hood, M.D.
1879 Edward Woakes, M.D.
Armand de Watteville, M.D.
Malcolm A. Morris.
A. E. Cumberbatch.
Edmund Owen.
Arthur E. J. Barker.
Frederick Treves.
Thomas John Maclagan, M.D.
Andrew Clark.
Francis Henry Champneys, M.D
William Watson Cheyne, F.R.S
George Henry Savage, M.D.

1879 H. H. Clutton, M.A.
Frederic S. Eve.
E. Noble Smith.
William Henry Allchin, M.D.
F. G. Dawtrey Drewitt, M.D.
1880 Robert Alex. Gibbons, M.D.
David Ferrier, M.D., F.R.S.
Vincent Dormer Harris, M.D.
Edmund Distin Maddick.
Jas. John MacWhirter Dunbar, M.D.
James William Browne, M.B.
William Appleton Meredith, M.B.
Malcolm Macdonald McHardy.
A. Boyce Barrow.
William Murrell, M.D.
Leslie Ogilvie, M.B.
George Ogilvie, M.B.
Charles Edward Beevor, M.D.
Thomas Colcott Fox, M.B.
George Henry Makins.
1881 Francis de Havilland Hall, M.D.
Robert Wharry, M.D.
Richard Clement Lucas, B.S.
Stephen Mackenzie, M.D.
William Hale White, M.D.
Eustace Smith, M.D.
Percy Kidd, M.D.
Oswald A. Browne, M.D.
W. Bruce Clarke, M.B.
Dawson Williams, M.D.
George Lindsay Johnson, M.D.
Henry Edward Juler.
Jonathan F. C. H. Macready.
C. B. Lockwood.
1882 Philip J. Hensley, M.D.
Ernest Clarke, M.D., B.S.
John Barclay Scriven.
George Robertson Turner.
Howard Henry Tooth, M.D.
Herbert Isambard Owen, M.D.
Charles R. B. Keetley.
Anthony A. Bowlby.
Amand J. McC. Routh, M.D.
Seymour J. Sharkey, M.D.
William Lang.
Henry Radcliffe Crocker, M.D.
1883 Edwin Clifford Beale, M.A., M.B.
James Kingston Fowler, M.D.
James Frederic Goodhart, M.D.
John Charles Galton, M.A.
W. Hamilton A. Jacobson, M.Ch.
Walter H. Jessop, M.B.
Walter Edmunds, M.C.
Victor A. Horsley, F.R.S.

1883 Dudley Wilmot Buxton, M.D.
Charles Douglas F. Phillips, M.D.
John James Pringle, M.B.
Henry Roxburgh Fuller, M.D.
Wilmot Parker Herringham, M.D.
Augustus Waller, M.D.
William Pasteur, M.D.
John Bland Sutton.
William Rose, M.B.
Storer Bennett.
Robert Marcus Gunn, M.B.
1884 George Newton Pitt, M.D.
Charles Stonham.
Stanley Boyd, M.B.
William Arbuthnot Lane, M.S.
Arthur Marmaduke Sheild, M.B.
Sidney Harris Cox Martin, M.D.,
F.R.S.
George Lawson.
Thomas Wakley, jun.
F. Swinford Edwards.
James Johnston, M.D.
William Duncan, M.D.
Charles Chinner Fuller.
Jean Samuel Keser, M.D.
George Richard Turner Phillips.
Bilton Pollard.
1885 Alexander Haig, M.D.
Theodore Dyke Acland, M.D.
Frederick Walker Mott, M.D.
James Berry.
John Cahill, M.D.
John Poland.
A. C. Butler-Smythe.
Charles Alfred Ballance, M.S.
Walter S. A. Griffith, M.D.
John Edward Squire, M.D.
John D. Malcolm, M.B., C.M.
Phineas S. Abraham, M.D.
Henry Willingham Gell, M.B.
1886 Robert Maguire, M.D.
Harrington Sainsbury, M.D.
Cuthbert Hilton Golding-Bird, M.B.
Lauriston Elgie Shaw, M.D.
Charters James Symonds, M.S.
Robert Boxall, M.D.
Allan Ogier Ward, M.D.
Archibald Edward Garrod, M.D.
Stephen Paget.
William Radford Dakin, M.D.
Samuel Herbert Habershon, M.D.
Arthur Quarry Silcock.
Arthur H. N. Lewers, M.D.
1887 Walter George Spencer.

1887 Thomas Outterson Wood, M.D.
Edgar William Willett, M.B.
Henry Lewis Jones, M.D.
Francis George Penrose, M.D.
Hugh Percy Dunn.
Frederic William Hewitt, M.D.
James Barry Ball, M.D.
Gilbert Richardson, M.D.
D'Arcy Power, M.B.
John Gay.
James Calvert, M.D.
Percy J. F. Lush, M.B.
1888 Robert Henry Scanes Spicer, M.D.
Jonathan Hutchinson, jun.
Campbell Williams.
James Donelan, M.B., C.M.
John Anderson, M.D., C.I.E.
Laurie Asher Lawrence.
Arthur Pearson Luff, M.D., B.Sc.
Albert Carless, M.S.
Frederick C. Wallis, M.B., B.C.
Charles James Cullingworth, M.D.
Edmund Cautley, M.D., B.C.
H. Montague Murray, M.D.
Arthur Symons Eccles, M.B.
Frank Joseph Wethered, M.D.
Edmund Wilkinson Roughton, B.S.
Frederick William Cock, M.D.
John Phillips, M.D.
1889 Montagu Handfield-Jones, M.D.
Norman M. MacLehose, M.B.
David Henry Goodsall.
Raymond Johnson, M.B.
John Fletcher Little, M.B.
Henry Work Dodd.
George Lindsay Turnbull, M.D.
Sidney Phillips, M.D.
William Charles Bull, M.B.
George P. Field.
John Wichenford Washbourn, M.D.
Charles Henry Cosens.
Henry Percy Dean, M.B., M.S.
Alfred Samuel Gubb, M.D.
William Hunter, M.D.
J. Inglis Parsons, M.D.
Bernard Pitts, M.B., M.C.
Robert Percy Smith, M.D., B.S.
Herbert R. Spencer, M.D., B.S.
Nestor Isidore Chas. Tirard, M.D.
1890 John Rose Bradford, M.D., F.R.S.
Roland Danvers Brinton. M.D.
Charles D. B. Hale, M.D.
Edwin Cooper Perry, M.D.
Morton Smale.

1890 Frederick Willcocks, M.D.
William T. Holmes Spicer, M.B.
Thomas Henry Crowle.
Henry Walter Syers, M.D.
Seymour Taylor, M.D.
William Alfred Wills, M.D.
G. O. White-Cooper, M.B.
Herbert William Allingham.
William Anderson.
William A. F. Bateman.
James Jackson Clarke, M.B.
Leonard G. Guthrie, M.D., B.Ch.
G. William Hill, M.D., B.Sc.
Edward Law, M.D., C.M.
Patrick Manson, M.D., C.M.
Humphry D. Rolleston, M.D.
Arthur Henry Ward.
Walter Essex Wynter, M.D., B.S.
1891 William Lee Dickinson, M.D.
Herbert P. Hawkins, M.D., B.Ch.
Cyril Ogle, M.A., M.B.
Arthur F. Voelcker, M.D., B.S.
Alfred Pownall Woodforde.
Herbert T. Herring, M.B., B.S.
Ernest Muirhead Little.
Henry Charrington Martin, M.D.
Frederick William Andrewes, M.D.
Alfred Eddowes, M.D.
Herbert Morley Fletcher, M.D.
William Heaton Hamer, M.D.
William Bromfield Paterson.
Holburt Jacob Waring.
Frederic Parkes Weber, M.D.
F. E. Batten, M.D.
Thomas Jessopp Bokenham.
Norman Dalton, M.D.
P. R. W. De Santi.
P. W. Dove.
William J. Gow, M.D.
Charles Arthur Mercier, M.B.
Paul Frank Moline, M.B.
Edward Percy Paton, M.D.
Arthur Bowen Rendel, M.B., B.C.
James Samuel Risien Russell,
    M.D.
Charles Percival White, M.B., B.C.
W. Page May, M.D.
Richard J. Reece.
1892 J. Dundas Grant, M.D.
R. J. Bliss Howard, M.D.
Thomas Horrocks Openshaw, M.B.
William Bezly Thorne, M.D.
W. H. Russell Forsbrook, M.D.
John Harold.

1892 John Alfred Masters, M.D.
     Gustave Schorstein, M.B.
     Charles Sempill de Segundo, M.B.
     John Tweedy.
     E. H. Myddelton-Gavey.
     E. Matthews James.
     J. S. Selwyn-Harvey, M.D.
     StClair Thomson, M.D.
     F. Manley B. Sims.
     F. Poynton Weaver, M.D.
     Henry Rayner, M.D.
     Walter S. Lazarus-Barlow, M.D.
     H. Marmaduke Page.
1893 James Taylor, M.D.
     Howard Barrett.
     Robert Cozens Bailey, M.B.
     Henry Albert Caley, M.D.
     Arthur Edward Giles, M.D.
     Miles Miley, M.B.
     D. Watkin Roberts, M.D.
     Leonard A. Bidwell.
     Frédéric F. Burghard, M.D., M.S.
     J. H. Drysdale, M.B.
     William McAdam Eccles, M.S.
     Vaughan Harley, M.D.
     George Herschell, M.D.
     Arnold Lawson.
     Guthrie Rankin.
     Walter Knowsley Sibley, M.D.
1894 Richard Gill.
     Joseph Sefton Sewill.
     Thomas Vincent Dickinson, M.D.
     Herbert Edward Durham, M.B.
     Alexander Morison, M.D.
     L. Hemington Pegler, M.D.
     Herbt. Furnivall Waterhouse, C.M.
     Percy Furnivall.
     R. L. Langdon-Down, M.B., B.C.
     Allan Macfadyen, M.D., B.S.
     Ernst Michels, M.D.
     Wm. Rivers Pollock, M.B., B.C.
     Charles Slater, M.B.
1895 Charles Arthur Parker.
     Sydney Russell Wells, M.D.
     Alfred Milne Gossage, M.B.
     Robert Murray Leslie, M.B.
     James Galloway, M.D.
     David Bridge Lees, M.D.
     Arthur G. Phear, M.D.
1896 Joseph Lockhart Downes, M.B.
     Edward Wilberforce Goodall, M.D.
     James Ernest Lane.
     George Oliver, M.D.
     George Alex. Sutherland, M.D.

1896 Charles Buttar, M.D.
     P. J. Freyer, M.D., I.M.S., M.A.
     Percival Horton-Smith, M.D.
     Frederick Henry Lewis, M.B.
     James Keogh Murphy, M.B.
     Thomas William Shore, M.D.
     John Stretton Sloane, M.B.
     William Aldren Turner, M.D.
     Arthur Nesham Weir, M.B.
     John Brian Christopherson, M.D.
     Charles Hubert Roberts, M.D.
     Charles R. J. Atkin Swan, M.B.
     James Kingston Barton.
     J. Walter Carr, M.D.
     John H. Dauber, M.A., M.B., B.Ch.
     Alexander Grant Russell Foulerton.
     L. Vernon Jones, B.A., M.D., B.Ch.
     Alexander MacGregor, M.D.
     Henry Betham Robinson, M.S.
     Horace George Turney, M.D.
     Ernest Waggett, M.B., B.C.
     Frederick Joseph Waldo, M.D.
     Hugh Walsham, M.D.
1897 Comyns Berkeley, M.B., B.C.
     William Arthur Brailey, M.D.
     James Cantlie, M.B.
     Raymond H. Payne Crawfurd, M.D.
     Louis Jenner, M.B.
     Charles Herbert Perram, M.D.
     Francis Whittaker Tunnicliffe, M.D.
     Arthur Whitfield, M.D.
     Arthur A. Jamison, M.D.
     Edward Stainer, M.A., M.B.
     Alfred G. Levy, M.D.
     A. P. Beddard, M.B.
     G. F. Blacker, M.D.
     W. S. Colman, M.D.
     F. W. Goodbody, M.D.
     R. Hutchison, M.D.
     Harold Low.
1898 J. H. Bryant, M.D.
     W. H. Corfield, M.D.
     L. A. Dunn, M.S.
     E. Hurry Fenwick.
     A. Downing Fripp, M.S.
     A. Corrie Keep, M.D.
     A. C. Latham, M.D.
     J. B. Lawford.
     John McFadyean.
     H. Murray Ramsay.
     J. F. H. Broadbent, M.D.
     H. Ronald Carter.
     A. Stark Currie, M.D.
     P. J. Edmunds, M.B.

1898 Alexander Granville.
James Morrison, M.D.
J. S. Edkins.
T. Jeeves Horder.
L. C. Powell Phillips.
F. W. Robertson.
S. Jervois Aarons, M.D.
Willmott Evans.
John Murray.
W. Adams Frost.
S. Backhouse Hulke.
C. R. C. Lyster.
Samuel Noble Bruce.
Cuthbert Chapman Gibbes, M.D.

1898 H. Stringfellow Pendlebury, M.B.
William Turner, M.B.
Alexander Crombie, M.D.
Thomas Herbert Kellock, M.D.
1899 Oswald Baker.
James Hugh Thursfield, M.B.
Lindley Marcroft Scott, M.D.
F. Rufenacht Walters, M.D.
Alfred P. Hillier, M.D.
Louis Bathe Rawling, M.B.
John Edward Sandiland, M.B.
Herbert Mundy.
Arthur J. Whiting, M.D.
W. H. Crosse.

The following Non-resident Fellows pay an annual subscription of £3 3s., and are thereby entitled to all the privileges of Resident Fellows.

Elected

1891   BRODIE, CHARLES GORDON, Fernhill, Wootton Bridge, Isle of Wight.

1888   CLARKE, ROBERT HENRY, M.B., New House, Mersham, Ashford, Kent.

1884   DRAGE, LOVELL, M.D., B.Ch.Oxon., Burleigh Mead, Hatfield, Herts.

1897   GILFORD, HASTINGS, Norwood House, King's road, Reading. *Trans.* 1.

1882   REID, THOMAS WHITEHEAD, M.D., Surgeon to the Kent and Canterbury Hospital; St. George's House, Canterbury.

1891   RUFFER, MARC ARMAND, The Quarantine Board, Alexandria.

1898   THOMAS, J. LYNN, Green-lawn, Pen-y-Lan, Cardiff.

NON-RESIDENT FELLOWS

1851 *Acland, Sir Henry W.*, Bart., K.C.B., M.D., LL.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Radcliffe Librarian, and late Regius Professor of Medicine in the University of Oxford; Oxford.

1897 *Addison, Christopher.* University College, Sheffield.

1866 *Allbutt, Thomas Clifford*, M.D., LL.D.Glasgow, F.R.S., Regius Professor of Physic, University of Cambridge; Consulting Physician to the Leeds General Infirmary; St. Rhadegund's, Cambridge. *Trans.* 3.

1884 *Anderson, Alexander Richard*, Surgeon to the General Hospital, 5, East Circus Street, Nottingham. *Trans.* 1.

1880 *Appleton, Henry*, M.D., 19, Regent terrace, Anlaby road, Hull.

1896 *Bagshawe, Frederic,* M.D., J.P., 35, Warrior Square, St. Leonard's-on-Sea.

1873 *Baker, J. Wright*, Consulting Surgeon to the Derbyshire General Infirmary [care of Dr. Benthall, 101, Friar gate, Derby]. Travelling.

1895 *Baldwin, Gerald R.*, 166, Victoria street, Melbourne, Australia.

1891 *Balgarnie, Wilfred*, M.B., The Dutch House, Hartley Wintney, Winchfield.

1896 *Ball, Charles Bent*, M.D., Ch.M., 24, Merrion square North, Dublin.

1866 *Banks, Sir John*, M.D., LL.D., D.Sc., K.C.B., Physician in Ordinary to the Queen in Ireland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Regius Professor of Physic in the University of Dublin; 45, Merrion square, Dublin.

Elected

1886   BANKS, SIR WILLIAM MITCHELL, M.D., Surgeon to the Liverpool Royal Infirmary; 28, Rodney street, Liverpool.

1882   BARKER, FREDERICK CHARLES, M.D., Surgeon-Major, Bombay Medical Service.

1881   Barnes, HENRY, M.D., LL.D., F.R.S. Ed., Physician to the Cumberland Infirmary; 6, Portland square, Carlisle.

1861   **Barnes**, ROBERT, M.D., Bernersmede, Eastbourne. C. 1877-8. V.P. 1889-90. *Referee*, 1867-76, 1891—. *Lib. Com.* 1869-73. *Sci. Com.* 1889—. *Trans.* 4.

1840   BARROW, BENJAMIN, Consulting Surgeon to the Royal Isle of Wight Infirmary; St. John's Lodge, Ryde.

1860   Bealey, ADAM, M.D., M.A., Filsham Lodge, Filsham road, St. Leonard's-on-Sea, Sussex.

1856   BEARDSLEY, AMOS, F.L.S., The Towers, Grange-over-Sands, Lancashire.

1896   BELBEN, FRANK, M.B., Endsleigh, Suffolk road, Bournemouth.

1880   BENNETT, ALEXANDER HUGHES, M.D. (Travelling).

1889   BENTLEY, ARTHUR J. M., M..D., Mena House, Pyramids, Cairo, Egypt.

1872   BEVERLEY, MICHAEL, M.D.. Consulting Surgeon to the Norfolk and Norwich Hospital; 54, Prince of Wales road, Norwich.

1865   Bickersteth, EDWARD ROBERT, Consulting Surgeon to the Liverpool Royal Infirmary; 2, Rodney street, Liverpool. *Trans.* 1.

1892   BICKERSTETH, ROBERT ALEXANDER, M.A., M.B., Assistant Surgeon to the Liverpool Royal Infirmary: 2, Rodney street, Liverpool.

1849   **Birkett**, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; Westbourne Rectory, Emsworth, Hampshire. C. 1865-6. *Referee*, 1851-9.

1865   BLANCHET, HILARION, 35, Conillard street, Quebec, Canada.

Elected

1890 BOSTOCK, *R. ASHTON*, Surgeon, Scots Guards, Cefn Mor, Penmaen, Glamorganshire.

1869 BOURNE, *WALTER*, M.D. (Travelling).

1874 BRADSHAW, *A. F.*, C.B., Surgeon Major-General, 111, Banbury road, Oxford.

1899 BRADSHAW, *THOMAS ROBERT*, M.D., 51, Rodney street, Liverpool. *Trans.* 2.

1876 BRIDGES, *ROBERT*, M.B., Manor House, Yattendon, Newbury, Berks.

1867 BRIDGEWATER, *THOMAS*, M.B., LL.D., Harrow-on-the-Hill, Middlesex.

1891 BRODIE, *CHARLES GORDON*, Fernhill, Wootton Bridge, Isle of Wight.

1892 BRONNER, *ADOLPH*, M.D., Senior Surgeon to Bradford Eye and Ear Hospital; Laryngologist to Bradford Royal Infirmary; 33, Manor row, Bradford.

1894 Brook, *WILLIAM HENRY BREFFIT*, M.D., B.S., 8, Eastgate, Lincoln.

1888 BROWNE, *HENRY LANGLEY*, Moor House, West Bromwich.

1881 BROWNE, *JOHN WALTON*, M.D., Surgeon to the Belfast Royal Hospital; Surgeon to the Belfast Ophthalmic Hospital; 10, College square N., Belfast.

1864 BUCKLE, *FLEETWOOD*, M.D., Merton Lodge, Merton road, Southsea.

1851 *Cadge, WILLIAM*, Consulting Surgeon to the Norfolk and Norwich Hospital; 49, St. Giles's street, Norwich. *Trans.* 1.

1891 CAMPBELL, *HENRY JOHNSTONE*, M.D., 36, Manningham lane, Bradford.

1888 CARTER, *WILLIAM JEFFREYS BECHER*, Aliwal North, Cape Colony.

1868 CAVAFY, *JOHN*, M.D., Consulting Physician to St. George's Hospital; 10, Fourth avenue, Hove, Sussex. C. 1887. *Referee*, 1896-9. *Lib. Com.* 1888-99. *Trans.* 1.

1898 *CAVE, EDWARD JOHN*, M.D., Bath.

Elected

1884　CHAFFEY, *WAYLAND CHARLES*, M.D., Physician to the Royal Alexandra Hospital for Children; 13, Montpellier road, Brighton.

1885　CHAPMAN, *PAUL MORGAN*, M.D., Physician to the Hereford General Infirmary, 1, St. John street, Hereford. *Trans.* 1.

1881　*Chavasse, THOMAS FREDERICK*, M.D., C.M., Senior Surgeon to the Birmingham General Hospital; 22, Temple row, Birmingham. *Trans.* 3.

1873　*Chisholm, EDWIN*, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.

1892　CLARK, *JAMES CHARLES*, 35, Castle road, Bedford.

1897　CLARK, *W. GLADSTONE*, 1, North road, Surbiton.

1888　CLARKE, *ROBERT HENRY*, M.B., New House, Mersham, Ashford, Kent.

1857　COATES, *CHARLES*, M.D., Consulting Physician to the Bath Royal United Hospital; 10, Circus, Bath.

1868　COCKLE, *JOHN*, A.M., M.D., Consulting Physician to the Royal Free Hospital; The Lodge, West Molesey. *Trans.* 2.

1893　COLE, *ROBERT HENRY*, M.D., Moorcroft, Hillingdon, Uxbridge.

1891　COOK, *HERBERT GEORGE*, M.D., B.S., 22, Newport road, Cardiff.

1891　COUMBE, *JOHN BATTEN*, M.D., Christchurch street, Ipswich.

1869　*Cresswell, PEARSON R.*, C.B., Senior Surgeon to the Merthyr General Hospital; Dowlais, Merthyr Tydfil.

1892　CROSS, *FRANCIS RICHARDSON*, M.B., Ophthalmic Surgeon to the Bristol Royal Infirmary, and Surgeon to the Bristol Eye Hospital; Worcester House, Clifton, Bristol.

1895　DARDEL, *JEAN*, M.D., Aix-les-Bains, Savoy.

1879　*DARWIN FRANCIS*, M.B., F.R.S., Wychfield, Huntingdon road, Cambridge.

Elected

1874 DAVIDSON, ALEXANDER, M.D., Consulting Physician to the Liverpool Royal Infirmary; Emeritus Professor, University College, Liverpool; 2, Gambier terrace, Liverpool.

1878 DAVY, RICHARD, Consulting Surgeon to the Westminster Hospital; Burstone House, Bow, North Devon. *Trans.* 1.

1882 DAWSON, YELVERTON, M.D., Heathlands, Southbourne-on-Sea, Hants.

1889 DELÉPINE, SHERIDAN, B.Sc., M.B., C.M., Professor of Pathology, Owens College, Manchester. *Trans.* 1.

1867 DRAGE, CHARLES, M.D., Hatfield, Herts.

1884 DRAGE, LOVELL, M.D.Oxon., Burleigh Mead, Hatfield, Herts.

1898 DRESCHFELD, JULIUS, Farndon House, Manchester.

1885 DRUMMOND, DAVID, M.D., 7, Saville place, Newcastle-on-Tyne.

1880 DRURY, CHARLES DENNIS HILL, M.D., Bondgate, Darlington.

1899 DRURY, EDWARD GUY DRU, M.B., B.S., Grahamstown, South Africa.

1871 DUKES, CLEMENT, M.D., B.S., Physician to Rugby School, and Senior Physician to the Hospital of St. Cross, Rugby; Sunnyside, Rugby, Warwickshire.

1867 DUKES, MAJOR CHARLES, M.D., Clarence Villa, Torrs park, Ilfracombe, North Devon.

1889 DUNCAN, JOHN, M.D., St. Petersburg, Russia.

1843 DURRANT, CHRISTOPHER MERCER, M.D., Consulting Physician to the East Suffolk and Ipswich Hospital; Northgate street, Ipswich, Suffolk.

1872 EAGER, REGINALD, M.D., Northwoods, near Bristol.

1887 EASMON, JOHN FARRELL, M.D., Assistant Colonial Surgeon, Gold Coast Colony, and Acting Chief Medical Officer of the Colony ; Accra, Gold Coast, West Africa.

1887 ELLIOTT, JOHN, 24, Nicholas street, Chester.

1848 ELLIS, GEORGE VINER, Minsterworth, Gloucester. C. 1863-4. *Trans.* 2.

Elected

1868　ELLIS, JAMES, M.D., The Sanatorium, Anaheim, Los Angeles County, California.

1889　ELLISTON, WILLIAM ALFRED, M.D., Stoke Hall, Ipswich.

1875　Fagan, JOHN, Consulting Surgeon to the Belfast Royal Hospital; 20, Fitzwilliam place, Dublin.

1897　FAGGE, THOMAS HENRY, M.D., Villa de la Porte Rouge, Monte Carlo.

1869　FAIRBANK, FREDERICK ROYSTON, M.D., Westcott, Dorking.

1872　Fenwick, JOHN C. J., M.D., Physician to the Durham County Hospital; Long Framlington, Morpeth.

1879　FINLAY, DAVID WHITE, M.D., Professor of the Practice of Medicine in the University of Aberdeen; Physician and Lecturer on Clinical Medicine to the Aberdeen Royal Infirmary; Consulting Physician to the Royal Hospital for Diseases of the Chest, London; 2, Queen's terrace, Aberdeen. *Referee*, 1891-3. *Trans.* 2.

1864　Folker, WILLIAM HENRY, Consulting and late Hon. Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.

1896　FORESTIER, HENRI, M.D., Aix-les-Bains, Savoie, France.

1892　FOSTER, MICHAEL GEORGE, M.A., M.B., Villa Annita, San Remo.

1896　FOULERTON, ALEXANDER GRANT RUSSELL, Dunsdale, Mulgrave road, Sutton, Surrey.

1859　FOX, EDWARD LONG, M.D., Consulting Physician to the Bristol Royal Infirmary; Church House, Clifton, Gloucestershire.

1884　Franks, KENDAL, M.D., Kilmurry, Hospital Hill, Johannesburg, South African Republic. *Trans.* 2.

1876　FURNER, WILLOUGHBY, M.D., Surgeon to the Sussex County Hospital; Brunswick square, Brighton.

Elected

1864 Gairdner, SIR WILLIAM TENNANT, M.D., K.C.B., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Western Infirmary, Glasgow ; 225, St. Vincent street, Glasgow. *Trans.* 1.

1885 GAMGEE, ARTHUR, M.D., F.R.S., Emeritus Professor of Physiology in the Owens College, Victoria University, Manchester; Montreux, Switzerland.

1867 GARLAND, EDWARD CHARLES, Yeovil, Somerset.

1879 GARSTANG, THOMAS WALTER HARROPP, Headingley House, Knutsford, Cheshire.

1889 Gaskell, WALTER HOLBROOK, M.D., F.R.S., Lecturer on Physiology, University of Cambridge; The Uplands, Great Shelford, Cambs.

1884 GIBBES, HENEAGE, M.D., Health Officer, Detroit, Michigan, U.S.A.

1897 GIBSON, GEORGE ALEXANDER, M.D., D.Sc., 17, Alva Street, Edinburgh.

1897 GILFORD, HASTINGS, Norwood House, King's road, Reading. *Trans.* 1.

1893 GORDON, WILLIAM, M.B., M.C.

1890 Gordon, WILLIAM, M.D., Barnfield Lodge, Exeter.

1898 GRAY, J. A., M.B., Wadham Lodge, Uxbridge road, Ealing.

1889 GREENE, GEORGE EDWARD JOSEPH, M.A., D.Sc., F.L.S., Monte Vista, Ferns, County Wexford.

1875 Greenfield, WILLIAM SMITH, M.D., Professor of Pathology and Clinical Medicine in the University of Edinburgh ; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee,* 1881.

1882 GRESSWELL, DAN ASTLEY, M.A., M.D., D.P.H., Chairman, Board of Public Health, Melbourne, Victoria.

1889 GRIFFITHS, JOSEPH, M.A., M.D., C.M., Reader in Surgery in the University of Cambridge ; Surgeon to Addenbrooke's Hospital; 63, Trumpington street, Cambridge. *Pro.* 1.

Elected

1870 HAMILTON, ROBERT, Consulting Surgeon to the Royal Southern Hospital, Liverpool; Magherabuoy, Portrush, Co. Antrim, Ireland.

1892 HARSANT, WILLIAM HENRY, Surgeon to the Bristol Royal Infirmary; The Tower House, Clifton road, Clifton, Bristol.

1854 HAVILAND, ALFRED, Ridgemount, Frimley Green, Surrey.

1890 HAVILAND, FRANK PAPILLON, M.D., B.C., 57, Warrior square, St. Leonard's-on-Sea.

1885 HAWKINS, FRANCIS HENRY, M.D., Physician to the Royal Berkshire Hospital; 73, London street, Reading. *Trans.* 1.

1860 Hayward, HENRY HOWARD, Consulting Surgeon Dentist to St. Mary's Hospital; Harbledown, 120, Queen's road, Richmond. C. 1878-9.

1861 HAYWARD, WILLIAM HENRY, Oxford road, Burnley, Lancashire.

1899 HIND, HENRY, Harrogate.

1843 Holden, LUTHER, Consulting Surgeon to St. Bartholomew's Hospital, Pinetoft, Ipswich. C. 1859. L. 1865. V.P. 1874. *Referee,* 1866-7. *Lib. Com.* 1858.

1894 HOLLAND, JAMES FRANK, M.D., St. Moritz, Engadine, Switzerland.

1868 HOLLIS, WILLIAM AINSLIE, M.D., Physician to the Sussex County Hospital ; 1, Palmeira avenue, Hove. *Trans.* 1.

1846 Holthouse, CARSTEN, Helidon House, Shoeburyness, Essex. C. 1863. *Referee,* 1870-6. *Lib. Com.* 1859-60.

1865 HOWARD, BENJAMIN, M.D. [New York, U.S.A.] *Trans.* 1.

1881 HOWARD, HENRY, M.B., Medical Officer of Health, Williamstown, Melbourne, Victoria.

1896 HUGHES, MATTHEW LOUIS, Capt., Royal Army Medical Corps [care of Messrs. Holt and Co., 17, Whitehall place, S.W.]. *Trans.* 1.

1882 HUMPHRY, LAURENCE, M.D., 3, Trinity street, Cambridge.

Elected

1896 HYDE, SAMUEL, M.D., Lismore House, 3, Hardwick street, Buxton.

1847 IMAGE, WILLIAM EDMUND, Herringswell House, Mildenhall, Suffolk. *Trans.* 1.

1863 JACKSON, THOMAS VINCENT, Surgeon to the Wolverhampton and Staffordshire General Hospital, &c.; Whetstone House, Waterloo road south, Wolverhampton.

1883 Jenkins, EDWARD JOHNSTONE, M.D., The Australian Club, Sydney, New South Wales.

1881 JENNINGS, WILLIAM OSCAR, M.D., 35, Rue Marbœuf, Avenue des Champs-Elysées, Paris.

1889 JOHNSON, HAROLD J., Senior Assistant, Gloucester County Asylum, Gloucester.

1848 JOHNSTONE, ATHOL ARCHIBALD WOOD, Consulting Surgeon to the Royal Alexandra Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Lib. Com.* 1860. *Trans.* 1.

1876 JONES, LESLIE HUDSON, M.D., Limefield House, Cheetham hill, Manchester.

1875 Jones, PHILIP SYDNEY, M.D., Consulting Surgeon to the Sydney Infirmary; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., Wool Exchange, Coleman Street, E.C.]

1865 JORDAN, FURNEAUX, Consulting Surgeon to the Queen's Hospital, Birmingham; Harborne, near Birmingham.

1872 KELLY, CHARLES, M.D., Ellesmere, Gratwicke road, Worthing, Sussex.

1848 Kendell, DANIEL BURTON, M.B., Thornhill House, Walton, near Wakefield, Yorkshire.

1890 Kerr, J. G. DOUGLAS, M.B., C.M., 6, The Circus, Bath.

1877 Khory, RUSTOMJEE NASERWANJEE, M.D.Brux., Hormazd Villa, Khumballa hill, Bombay.

Elected

1898 *KLEFSTAD-SILLONVILLE, O.,* M.D., Aix-les-Bains, Savoie.

1888 *KYNSEY, SIR WILLIAM RAYMOND,* C.M.G., Oriental Club, Hanover square. (Travelling.)

1889 *LANCASTER, ERNEST LE CRONIER,* M.B., B.Ch., Assistant Physician to the Swansea Hospital; Hon. Physician to the Swansea and South Wales Institution for the Blind; Winchester House, Swansea, S. Wales.

1873 *Larcher, O.,* M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.

1862 *LATHAM, PETER WALLWORK,* M.D., Downing Professor of Medicine, Cambridge University, 1874-94; Senior Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.

1890 *Lawrie, EDWARD,* M.B., Surgeon Lieutenant - Colonel, Indian Medical Department; Residency Surgeon; Hyderabad, Deccan.

1880 *LAYCOCK, GEORGE LOCKWOOD,* M.B., C.M., Melbourne, Victoria, Australia.

1886 *Lediard, HENRY AMBROSE,* M.D., Surgeon to the Cumberland Infirmary; 35, Lowther street, Carlisle. *Trans.* 1.

1882 *LEDWICH, EDWARD L'ESTRANGE,* Anatomist to the Royal College of Surgeons, Ireland; 30, Upper Fitzwilliam street, Dublin.

1895 *LEECH, DANIEL JOHN,* M.D., Elm House, Whalley Range, Manchester. *Sci. Com.* 1896—.

1883 *LEESON, JOHN RUDD,* M.D., C.M., Clifden House, Twickenham.

1869 *LEGG, JOHN WICKHAM,* M.D. C. 1886. *Referee,* 1882-5. *Lib. Com.* 1878-85. *Trans.* 2.

1898 *LINDSAY, JAMES,* M.A., M.D., 13, College square east, Belfast.

1872 *Little, DAVID,* M.D., Senior Surgeon to the Royal Eye Hospital, Manchester; Ophthalmic Surgeon to the Manchester Royal Infirmary; Lecturer on Ophthalmology at the Victoria University; 21, St. John street, Manchester.

Elected

1889 *Little, James*, M.D., Physician to the Adelaide Hospital; 14, Stephen's Green North, Dublin.

1871 *Little, Louis Stromeyer*, Shanghai, China.

1894 *Lowe, Thomas Pagan*, 16, The Circus, Bath.

1867 *Maberly, George Frederick*, Mailai Valley, Nelson, New Zealand.

1889 *MacAlister, Donald*, M.A., B.Sc., M.D., Physician to Addenbrooke's Hospital; Linacre Lecturer and Tutor, St. John's College; University Lecturer in Medicine; St. John's College, Cambridge.

1887 *Macdonald, George Childs*, M.D.

1866 *Macgowan, Alexander Thorburn*, M.D., Vyvian House, Clifton park, Bristol.

1859 *M'Intyre, John*, M.D., LL.D., Odiham, Hants.

1876 *Mackey, Edward*, M.D., Physician to the Sussex County Hospital; Senior Physician to the Royal Alexandra Hospital for Sick Children; 56, Lansdowne place, Brighton.

1854 *Mackinder, Draper*, M.D., 26, Denmark Villas, Hove, Sussex.

1893 *MacLeod, Surgeon-Colonel Kenneth*, M.D., The Towers, Woolston, S. Hants.

1891 *Manby, Alan Reeve*, M.D., Surgeon Apothecary to their Royal Highnesses the Prince and Princess of Wales and to the Duke and Duchess of York at Sandringham; East Rudham, Norfolk.

1894 *Marriott, Charles William*, M.D., Aubrey House, Bath road, Reading.

1892 *Martin, Christopher*, M.B., C.M., Surgeon to the Birmingham and Midland Hospital for Women; 35, George road, Edgbaston, Birmingham.

1899 *Martyn, Gilbert John King*, M.D., 12, Gay street, Bath.

1883 *Maudsley, Henry Carr*, M.D., 22, Collins street, Melbourne, Victoria.

Elected

1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1897 MERRY, WILLIAM JOSEPH COLLINGS, M.D, B.Ch., 2, Chiswick place, Eastbourne.

1898 Millard, WILLIAM JOSEPH KELSON, M.D., 7, Bayshill villas, Cheltenham.

1895 MILLS-ROBERTS, ROBERT HERBERT, Hafod-ty, Llanberis, North Wales.

1887 MIVART, FREDERICK ST. GEORGE, M.D., Local Government Board Inspector; 6, Edge hill, Wimbledon.

1896 MOORE, JOHN WILLIAM, M.D., M.Ch., 40, Fitzwilliam square west, Dublin.

1891 MORRIS, GRAHAM, Wallington, Surrey.

1894 MORSE, THOMAS HERBERT, All Saints' Green, Norwich. *Trans.* 1.

1873 MURRAY, J. IVOR, M.D., F.R.S.E., Granby House, Scarborough.

1881 NALL, SAMUEL, M.B., Dryhurst Lodge, Disley, Stockport.

1889 NAPIER, FRANCIS HORATIO, M.B., Cape Town.

1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine and Psychological Medicine in the University of Melbourne; 21, Spring street, Melbourne, Victoria.

1895 NEWSHOLME, ARTHUR, M.D., 11, Gloucester place, Brighton.

1868 NICHOLLS, JAMES, M.D., Trekenning House, St. Columb, Cornwall.

1849 NORMAN, HENRY BURFORD, The Manor-house, Drayton, Taunton, Somerset. *Lib. Com.* 1857.

1847 Nourse, WILLIAM EDWARD CHARLES, Norfolk Lodge, Thurloe road, Torquay.

1884 OAKES, ARTHUR, M.D., Narrabri, Cole Park road, Twickenham.

1880 O'CONNOR, BERNARD, A.B., M.D., Senior Physician to the North London Hospital for Consumption; 25, Hamilton road, Ealing.

1896 OGLE, JOHN GILBERT, M.D., South Redlands, Reigate.

1855 Ogle, WILLIAM, M.A., M.D., Consulting Physician to the Royal Derbyshire Infirmary; The Elms, Duffield road, Derby.

Elected

1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].

1883 *Oliver, THOMAS,* M.A., M.D., Professor of Physiology, University of Durham; and Physician to the Newcastle-on-Tyne Infirmary; 7, Ellison place, Newcastle-on-Tyne. *Trans.* 1.

1871 *O'Neill, WILLIAM,* M.D., C.M., late Physician to the Lincoln Lunatic Hospital, and Physician Lincoln General Dispensary, &c.; 2, Lindum road, Lincoln.

1890 ORD, WILLIAM WALLIS, M.D., The Hall, Salisbury.

1885 ORMSBY, L. HEPENSTAL, M.D., Lecturer on Clinical and Operative Surgery and Surgeon to the Meath Hospital and County Dublin Infirmary; Surgeon to the Children's Hospital, Dublin; 92, Merrion square west, Dublin.

1887 PAGET, CHARLES EDWARD, Medical Officer of Health to the County Council of Northamptonshire; County Hall, Northampton.

1858 *Paley, WILLIAM,* M.D., Physician to the Ripon Dispensary; Yore Bank, Ripon, Yorkshire.

1887 PARDINGTON, GEORGE LUCAS, M.D., 47, Mount Pleasant road, Tunbridge Wells.

1885 PARKER, RUSHTON, M.B., B.S., Professor of Surgery, University College, Liverpool (Victoria University); Surgeon to the Liverpool Royal Infirmary; 59, Rodney street, Liverpool.

1891 PARKIN, ALFRED, M.S., M.D., 24, Albion street, Hull. *Trans.* 1.

1879 PEEL, ROBERT, 120, Collins street east, Melbourne, Victoria.

1874 PENHALL, JOHN THOMAS, The Cedars, Broadwas-on-Teme, Worcester.

1879 *Pesikaka, HORMASJI DOSABHAI,* 43, Hornby road, Bombay.

1878 *Philipson, GEORGE HARE,* M.D., D.C.L., Professor of Medicine in Durham University; Consulting Physician to the Newcastle-upon-Tyne Royal Infirmary; 7, Eldon square, Newcastle-upon-Tyne.

Elected

1891 PIERCE, BEDFORD, M.D., The Retreat, York.

1897 PIGG, T. STRANGEWAYS, 62, Jesus Lane, Cambridge.

1841 **Pitman,** *SIR HENRY ALFRED*, M.D., Consulting Physician
to St. George's Hospital ; Cranbrook, Bycullah park,
Enfield. L. 1851-3. C. 1861-2. T. 1863-8. V.P.
1870-1. *Referee,* 1849-50. *Lib. Com.* 1847.

1892 POWELL, HERBERT ANDREWS, M.A., M.D., M.Ch., Piccards
Rough, Guildford.

1898 PRENDERGAST, VINCENT, 9, Rue Volney, Paris.

1897 QUARTEY-PAPAFIO, BENJAMIN WILLIAM, M.D., Accra,
Gold Coast, West Africa.

1857 VON RANKE, HENRY, M.D., 3, Sophienstrasse, Munich.

1890 RANSOM, WILLIAM BRAMWELL, M.D., Physician to the
Nottingham General Hospital; The Pavement, Not-
tingham. *Trans.* 1.

1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Consulting
Physician to the Nottingham General Hospital; 17,
Park Valley, Nottingham. *Trans.* 1.

1882 REID, SIR JAMES, Bart., M.D., K.C.B., Resident Physi-
cian and Physician in Ordinary to H.M. the Queen,
Windsor Castle.

1884 REID, THOMAS WHITEHEAD, M.D., Surgeon to the Kent
and Canterbury Hospital ; St. George's House, Canter-
bury, Kent.

1881 RICE, GEORGE, M.B., C.M., Sutton, Surrey.

1889 RIVERS, W. H. RIVERS, M.D., St. John's College, Cam-
bridge.

1871 Roberts, DAVID LLOYD, M.D., F.R.S.E., Consulting Obstet-
ric Physician to the Manchester Royal Infirmary ; Phy-
sician to St. Mary's Hospital, and Lecturer on Clinical
Obstetrics and Gynæcology at the Owens College,
Manchester ; 11, St. John street, Manchester.

1889 ROBERTS, LESLIE, M.D., 46, Rodney street, Liverpool.

1873 ROBERTSON, WILLIAM HENRY, M.D., Consulting Physician
to the Buxton Bath Charity and Devonshire Hospital ;
Buxton, Derbyshire.

1888 Robinson, FREDERICK WILLIAM, M.D., C.M., Huddersfield.

Elected

1889 ROBSON, ARTHUR WILLIAM MAYO, Professor of Surgery, Yorkshire College; Senior Surgeon, Leeds General Infirmary; 7, Park square, Leeds. *Trans.* 4. *Pro.* 1.

1885 ROCKWOOD, WILLIAM GABRIEL, M.D., Colombo, Ceylon.

1898 ROGERS, LEONARD, I.M.S. [care of Messrs. Watson & Co., Bombay]. *Trans.* 2.

1889 ROSS, DANIEL McCLURE, M.D., Cedar Lodge, Littledown Road, Bournemouth.

1863 ROWE, THOMAS SMITH, M.D., Consulting Surgeon to the Royal Sea-Bathing Infirmary; Union crescent, Margate, Kent.

1891 RUFFER, MARC ARMAND, M.D., The Quarantine Board, Alexandria.

1898 SALTER, A., M.D., The Poplars, Sudbury, Harrow.

1855 **Sanderson,** SIR JOHN BURDON, BART., M.D., LL.D., D.C.L.Durham, D.Sc., F.R.S., Regius Professor of Medicine in the University of Oxford; 64, Banbury road, Oxford. C. 1869-70. V.P. 1882. *Referee,* 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.

1867 SANDFORD, FOLLIOTT JAMES, M.D., V.D., late Surgeon-Major, 2nd Batt. S.V.L.Infy., now Hon. Surgeon-Major; Surgeon to the Market Drayton Dispensary, and Consulting Physician to the Market Drayton Cottage Hospital ; Market Drayton, Shropshire.

1886 SAUNDBY, ROBERT, M.D., LL.D., Physician to the General Hospital, and Consulting Physician to the Hospital for Women, and to the Eye Hospital, Birmingham ; Professor of Medicine, Mason University College; 83A, Edmund street, Birmingham.

1891 SAUNDERS, FREDERICK WILLIAM, M.B., B.C., Chieveley House, near Newbury, Berks.

1883 SCHAFER, EDWARD ALBERT, LL.D., F.R.S., Professor of Physiology in the University of Edinburgh. C. 1899—. *Referee,* 1888-99. *Sci. Com.* 1889—.

1861 SCOTT, WILLIAM, M.D., Senior Physician to the Huddersfield Infirmary ; Waverley House, Huddersfield.

1897 SEMPLE, EDWARD, M.D., Grove house, Fenstanton, Hunts.

Elected

1897 SEYMOUR, SURG.-MAJOR CHARLES, Bareilly, North-West Provinces, India.

1899 SHUTTLEWORTH, GEORGE EDWARD, M.D., Ancaster House, Richmond Hill.

1887 SIDEBOTHAM, EDWARD JOHN, M.B., Erlesdene, Bowdon, Cheshire.

1857 SIORDET, JAMES LEWIS, M.B., Villa Cabrolles, Mentone, Alpes Maritimes, France.

1891 SMITH, G. COCKBURN, M.D., 29, Lansdown crescent, Cheltenham.

1886 SMITH, HOWARD LYON, Buckland House, Buckland Newton, Dorchester.

1894 SMITH, ROBERT SHINGLETON, M.D., B.Sc., Deepholm, Clifton Park, Clifton, Bristol.

1894 SMITH, THOMAS RUDOLPH, M.B., B.C., Blytheholm, Stockton-on-Tees.

1868 SOLLY, SAMUEL EDWIN, Colorado Springs, Colorado, U.S.A.

1899 STEPHEN, GUY NEVILLE, Foreign Office Medical Staff.

1896 STEPHENS, JOHN WILLIAM WATSON, M.B., B.C., Pathological Laboratory, Cambridge.

1891 STEVENS, SURG.-CAPT CECIL ROBERT, M.B., B.S., I.M.S., Eden Hospital, Calcutta.

1854 STEVENS, HENRY, M.D., late Inspector, Medical Department, Local Government Board, Whitehall; Durham Lodge, St. Margaret's road, Twickenham.

1884 STEWART, EDWARD, M.D., Brook House, East Grinstead.

1879 Stirling, EDWARD CHARLES, M.D., Senior Surgeon to the Adelaide Hospital; Lecturer on Physiology in the University of Adelaide, South Australia [care of Messrs. Elder and Co., 7, St. Helen's place].

1865 STOKES, SIR WILLIAM, M.D., M.C., Surgeon to the Meath Hospital; 5, Merrion square north, Dublin. *Trans.* 1.

1871 STRONG, HENRY JOHN, M.D., J.P., Consulting Surgeon to the Croydon General Hospital; Colonnade House, The Steyne, Worthing.

Elected

1890 *Sympson, E. Mansel,* M.D., B.C., Surgeon to the Lincoln County Hospital; Deloraine Court, Lincoln.

1886 *Teale, Thomas Pridgin,* M.B., F.R.S., Consulting Surgeon to the Leeds General Infirmary; 38, Cookridge street, Leeds.

1898 *Thomas, J. Lynn,* Green Lawn, Pen-y-lan, Cardiff.

1890 *Thomas, William Robert,* M.D., Little Forest, Bath road, Bournemouth.

1891 *Thomson, John Roberts,* M.D., Monkchester, Bournemouth.

1876 *Thornton, J. Knowsley,* M.B., C.M., Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Hildersham Hall, Cambridge. C. 1891. *Lib. Com.* 1886-90, 1893-95. *Trans.* 5.

1883 *Thursfield, Thomas William,* M.D., Physician to the Warneford and South Warwickshire General Hospital; Selwood, Beauchamp square, Leamington.

1880 *Tivy, William James,* 8, Lansdowne place, Clifton, Bristol.

1871 *Trend, Theophilus W.,* M.D., 1, Grosvenor square, Southampton.

1881 *Treves, William Knight,* Surgeon to the National Hospital for Scrofula; 31, Dalby square, Cliftonville, Margate.

1867 *Trotter, John William,* formerly Surgeon-Major, Coldstream Guards; 4, St. Peter's terrace, York.

1873 *Turner, George Brown,* M.D., Camden House, Hemel Hempsted, Herts.

1894 *Turner, Philip Dymock,* M.D. (Travelling).

1891 *Tweed, Reginald,* M.D., Higher Leyhill, Hembury Fort, Honiton.

Elected

1881   TYSON, WILLIAM JOSEPH, M.D., Senior Medical Officer of the Victoria Hospital, Folkestone; 10, Langhorne Gardens, Folkestone.

1854   WADDINGTON, EDWARD, Hamilton, Auckland, New Zealand.

1868   Walker, ROBERT, Clovelly, Bideford.

1887   WALLACE, EDWARD JAMES, M.D., Holmbush, Grove road, Southsea.

1867   WALLIS, GEORGE, Consulting Surgeon to Addenbrooke's Hospital; 6, Hills road, Cambridge.

1883   Walters, JAMES HOPKINS, Surgeon to the Royal Berkshire Hospital; 15, Friar street, Reading.

1846   WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.

1861   WATERS, A. T. HOUGHTON, M.D., Consulting Physician to the Royal Infirmary; 69, Bedford street, Liverpool. *Trans.* 3.

1874   WELLS, HARRY, M.D., San Ysidro, Buenos Ayres, S. America.

1882   WHARRY, CHARLES JOHN, M.D., 14, Ewell road, Surbiton, Surrey.

1897   WHITE, CHARLES POWELL, 130, Hyde Park road, Leeds.

1881   Whitehead, WALTER, F.R.S. Ed., Senior Surgeon to the Manchester Royal Infirmary, Manchester and Salford Lock Hospital, and Manchester and Salford Skin Hospital; Professor of Clinical Surgery, Owens College, Victoria University; 499, Oxford road, Manchester. *Trans.* 1.

1885   Whitla, WILLIAM, M.A., M.D., Professor of Materia Medica and Therapeutics, Queen's College, Belfast; Physician to, and Lecturer in Medicine at, the Belfast Royal Hospital; Consulting Physician to the Ulster Hospital for Women and Children; Consulting Physician to the Belfast Ophthalmic Hospital; 8, College square north, Belfast.

Elected

1852 *WIBLIN, JOHN,* The Hermitage, Clewer, Windsor. *Trans.* 1.

1870 *Wilkin, JOHN F.,* M.D., M.C., Rose Ash House, South Molton.

1883 *Willans, WILLIAM BLUNDELL,* Much Hadham, Herts.

1896 *WILLIAMS, ALFRED HENRY,* M.D., Rotorua, Harrow.

1859 *Williams, CHARLES,* Senior Surgeon to the Norfolk and Norwich Hospital; 48, Prince of Wales road, Norwich.

1887 *WILSON, ARTHUR HERVEY,* M.D., 504, Broadway, Boston, U.S.A.

1889 *WISE, A. TUCKER,* M.D., Montreux, Switzerland.

1850 *Wise, ROBERT STANTON,* M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Beech Lawn, Banbury.

1885 *WOLFENDEN, RICHARD NORRIS,* M.D., Rangemont, Seaford, Sussex.

1892 *WOODHEAD, GERMAN SIMS,* M.D., Professor of Pathology in the University of Cambridge; 6, Scrope terrace, Cambridge.

1879 *WOODWARD, G. P. M.,* M.D., Deputy Surgeon-General; 157, Liverpool street, Hyde Park, Sydney, New South Wales.

1892 *WRIGHT, ALMROTH EDWARD,* M.D., Ch.B., Oakhurst, Netley, Hants.

ANNUAL MEETING,

March 1st, 1899, at 5 p.m.

THOMAS BRYANT, President, in the Chair.

NORMAN MOORE, M.D., } Hon. Secs.
A. PEARCE GOULD, M.S., }

The Minutes of the last Annual Meeting were read and signed.

The President nominated Dr. Rolleston and Mr. Venning as Scrutineers, and declared the Ballot open until six p.m.

Dr. Moore (Hon. Secretary) read the Report of the Council.

REPORT OF COUNCIL.

THE Council has to report that the affairs of the Society are in a satisfactory condition, and that numerous and important papers have been submitted to the meetings. Thirty-five new Fellows have been elected, of whom 24 are Resident and 11 Non-resident. The Council have heard with regret since the last Annual Meeting of the deaths of 5 Resident and 4 Non-resident Fellows. Three Fellows have resigned. The following Reports have been received and are appended :

Report of the Committee on Suspended Animation.

Report of the Committee on Climates and Baths of the United Kingdom.

Report of the Honorary Treasurers.

Report of the Honorary Librarians.

It is believed that the arrangements which came into force last year with regard to the Library hours have been found to add to the usefulness of the Society to its Fellows.

During the absence of the Resident Librarian, in accordance with the special vote of the Council on account of his ill-health, the services of Mr. Clarke, the Sub-Librarian, deserve special commendation.

The Society now contains 16 Honorary Fellows, 527 Resident Fellows, 278 Non-resident Fellows.

REPORT OF THE SUSPENDED ANIMATION COMMITTEE.

" The Suspended Animation Committee have arranged to have some experiments carried out at Claybury on the dead subject. Until these are completed they have nothing further to report."

REPORT OF THE COMMITTEE ON CLIMATES AND BATHS.

" No Report has been received since February 23rd, 1898, when Dr. Ewart read his report on Middlesex and London. Progress has, however, been made with the remaining work. The Reports on North Wales (Dr. Leech) and on the East Coast (Dr. Murrell) are nearly ready, while it is hoped that those on Ireland and the Midlands will also be ready during the current year."

TREASURERS' REPORT.

" The Treasurers desire to draw attention to the altered form in which the accounts are this year presented to the Society.

"Prior to the removal of the Society from Berners Street to its present premises, a statement of the yearly receipts and payments sufficed to show its financial condition; its income was then derived from the contributions of the Fellows, and its expenditure was confined to the objects of the Society and the maintenance of its House and Library. The position is now different, as the Society possesses valuable property producing a large income, while at the same time a large proportion of its total income has to be set aside for the payment of interest and the gradual reduction of its debenture debt.

"The Treasurers have for some years felt that the accounts should be presented in such a form that the Fellows might be able to see year by year the true financial position. Two years ago, the property of the Society having to be re-valued on the conversion of the debenture debt, the Treasurers took that opportunity of introducing into the yearly accounts a statement of the Liabilities and Assets of the Society, and they have now added to the statement of the receipts and payments a complete revenue account showing the exact financial position of the Society on December 31st, 1898.

"The Treasurers believe that with the auditor's assistance there will be no difficulty in continuing to keep the accounts in their present form, and they wish to express their thanks to Mr. Mundy for the valuable assistance he has given them, and for the great amount of work that he has done in rearranging the books and preparing the revenue account.

"The Society can be congratulated on its present financial position. During the year £400 of debentures have been paid off, and the floating debt has been reduced by £163 1s. 11d."

Honorary Librarians' Report.

"The work of the Library has increased, and it is now more used by Fellows than ever before. During the past year 3493 volumes have been issued to Fellows, and 216 volumes have been borrowed from Mr. Lewis's Library to supply the immediate demands of Fellows.

"By purchase, 351 volumes have been added to the Library, exclusive of various 'Transactions,' &c., in continuation.

"The Library has been enriched by donations of 241 books and pamphlets (exclusive of 'Transactions,' &c.). The total additions for the year are 592."

The Council appointed a Committee consisting of the President, the Hon. Secretaries, Dr. Allchin, Dr. Rose Bradford, Mr. Eve, Dr. Ferrier, Dr. Hughlings Jackson, Mr. Jacobson, and Dr. Savage, to report on the award of the Marshall Hall Prize "for the best original work done during the previous five years and recorded in the English language, in anatomical, physiological, or pathological research relative to the nervous system." The Committee unanimously recommended the award of the prize to Dr. C. S. Sherrington, and in accordance with their report the Council recommend the award of the prize by the Society to him.

Dr. Church (Hon. Treasurer) explained the accounts.

The President moved—"That the Report of the Council, together with the Treasurers' Statement of Accounts, be adopted." Carried.

The President moved—"That the Marshall Hall Prize be awarded to Dr. Charles Scott Sherrington." Carried.

The PRESIDENT, addressing himself to Dr. Sherrington, said he had the greatest pleasure in greeting him as the recipient of the Marshall Hall Prize, a prize given by the Society on the recommendation of the Council and the advice of a special committee constituted for that purpose. He warmly congratulated him on behalf of the Council and Fellows of the Society upon his success. He expressed the hope that the Society might at one of its early meetings hear from Dr. Sherrington's lips some account of the good work he had done and for which the prize had been awarded. The President then presented Dr. Sherrington with the Marshall Hall Prize, being a cheque for £81 19s. 8d. and the diploma recording the award.

Professor SHERRINGTON expressed his profound appreciation of the honour which the Society had done him in making this award. He could only wish that the researches which they had so liberally appreciated in this way had been a little more worthy of the honour they were receiving. The association with the name of Marshall Hall was one which enhanced the value of the distinction in the eyes of every neurologist. The name of Marshall Hall, indeed, required no additional stimulus to keep it alive among those who followed that particular branch of experimentation and knowledge. He added that he felt a very peculiar satisfaction at receiving this recognition of his imperfect researches. It was a sort of earnest that the association between laboratory and clinical work was not so likely to be divorced and separated as one might suppose. Physiologists and clinicians generally pursued separate paths, although there were brilliant exceptions in the bosom of that very society. He had received that kind of encouragement which would make him feel that his work had not been altogether unfruitful to clinicians.

The PRESIDENT then read the

ANNUAL ADDRESS (p. ci).

Dr. DICKINSON moved, and Dr. BOWLES seconded—"That the thanks of the Society be given to the President for his Address, and that he be requested to allow it to be printed in the 'Transactions.'" Carried.

Dr. BLANDFORD moved, and Mr. GOULD seconded—"That the thanks of the Society be given to the retiring Vice-Presidents, Dr. Marcet and Mr. Gant, for their services to the Society during their respective terms of office." Carried.

Dr. CHURCH moved, and Dr. ROLLESTON seconded—"That the thanks of the Society be given to the retiring Hon. Secretary, Dr. Norman Moore, for his services to the Society during his term of office." Carried.

Dr. CHAMPNEYS moved, and Mr. HAWARD seconded—"That the best thanks of the Society be given to Dr. Samuel J. Gee for his valuable services to the Society as Hon. Librarian since July, 1887." Carried.

Mr. HEATH moved, and Dr. WILLIAMS seconded—"That the thanks of the Society be given to the retiring Members of the Council, Dr. Allchin, Dr. Bowles, Dr. Mitchell Bruce, Dr. Savage, Mr. Watson Cheyne, Mr. Clutton, Mr. Frederic Eve, and Mr. Meredith, for their services to the Society during their respective terms of office." Carried.

The Scrutineers reported that the ballot had been duly held and that the following candidates had been elected:

President.—Thomas Bryant.

Vice-Presidents.—George Fielding Blandford, M.D.; Samuel Jones Gee, M.D.; Henry Greenway Howse, M.D.; Reginald Harrison.

Honorary Treasurers.—William Selby Church, M.D.; J. Warrington Haward.

Honorary Secretaries.—Thomas Barlow, M.D.; A. Pearce Gould, M.S.

Honorary Librarians.—Norman Moore, M.D.; Rickman J. Godlee, M.S.

Members of Council.—Francis Henry Champneys, M.D.; Stephen Mackenzie, M.D.; Seymour John

Sharkey, M.D.; Eustace Smith, M.D.; Francis Warner, M.D.; W. Bruce Clarke; George Henry Makins; Robert William Parker; Edward Albert Schäfer; Edgcombe Venning.

STATEMENT OF CASH RECEIPTS AND PAYMENTS FOR THE YEAR ENDING 31ST DECEMBER, 1898

Receipts

	£ s. d.	£ s. d.	£ s. d.
Balance 1st January, 1898 ...			127 4 7
Subscriptions, Fees, &c.:			
Annual Subscriptions at £3 3s....	1368 19 0		
Annual Subscriptions at £1 1s.	66 3 0		
Composition Fees (Life)	121 16 0		
Entrance Fees	226 16 0		
		1778 14 0	
'Transactions' and 'Proceedings:'			
Sold by Messrs. Longman	50 12 10		
„ Mr. H. K. Lewis	2 7 3		
„ Resident Librarian	15 15 8		
		68 15 9	
Rents		2643 19 3	
Interest:			
On Permanent Endowment Fund	12 12 4		
Miscellaneous Receipts ...	2 12 5		
		4506 13 9	
Permanent Endowment Fund:			
Donation		5 5 0	
			£4689 8 4

Payments

	£ s. d.	£ s. d.	£ s. d.
Rent, Rates, &c.	286 18 0		
Lighting, Cleaning, &c.	315 11 1		
Repairs, Alterations Furniture, &c.	54 10 5		
Meeting Expenses	24 13 11		
Printing, Stationery, Stamps, Telegrams, &c....	120 3 2		
Printing of Catalogue Supplement	70 14 3		
			872 5 10
Officers and Servants:			
Salaries and Wages ...	889 12 10		
Library:—Books and Binding ...	342 4 6		
'Transactions' and 'Proceedings'	586 5 7		
Debentures:			
Four Bonds redeemed... ...	400 0 0		
Interest	1021 10 0		
			3189 12 11
Auditors' Fee			10 10 0
Macmillan & Co., for 'Climates and Baths'			142 1 9
Miscellaneous			88 8 6
Balance at Bank and in hand			336 4 4
			£4689 8 4

(B)   Income and Expenditure Account for the Year ending 31st December, 1898

Expenditure

	£ s. d.	£ s. d.
Rent, Rates, and Taxes	266 14 7	
Salaries and Wages	839 12 10	
Lighting, Warming, and Cleaning ..	303 0 11	
Printing and Stationery, Stamps and Telegrams	162 4 0	
Meeting Expenses	24 13 11	
Miscellaneous Disbursements ..	91 11 1	
		1687 17 4
Repairs		62 3 0
Interest on Debentures		1055 14 8
Depreciation of Library Purchases ..	130 6 7	
Depreciation of Fixtures, Fittings, &c. ..	67 16 0	
		198 2 7
'Transactions,' &c.	485 12 2	
Less Amount of Sales	68 15 9	
		416 16 5
Audit Fee		10 10 0
Balance, being excess of Income over Expenditure during the year ..		762 9 3
		£4193 13 3

Income

	£ s. d.	£ s. d.
433 Annual Subscriptions at £3 3s. ..	1363 19 0	
63 Do. £1 1s. ..	66 3 0	
		1430 2 0
Composition Fees		121 16 0
Entrance Fees		226 16 0
Rents Receivable		2399 14 6
Interest on New South Wales Stock ..		12 12 4
Miscellaneous Receipts		2 12 5
		£4193 13 3

10th February, 1899.

Audited and found correct,
TOM MUNDY,
Chartered Accountant.

(C)

STATEMENT OF LIABILITIES AND ASSETS, 31st DECEMBER, 1898

LIABILITIES.

	£	s.	d.
3 per Cent. First Mortgage Debentures .	34,900	0	0
[This amount has been reduced by £400 since the previous statement by cash paid in respect to Debentures redeemed.]			
Sundry Creditors . . .	1,073	4	1
Endowment Fund Account .	5	5	0
Balance, being surplus of Assets over Liabilities	26,131	12	9
[Surplus, 31st December, 1897 £25,679 11 9			
Deduct—			
Liabilities not included . £214 12 1			
Items charged against the surplus on the adjustment of the accounts . 95 16 2			
	310	8	3
	25,369	3	6
Add—			
Excess of Income over Expenditure, 1898 .	762	9	3
	£26,131	12	9]
	£62,110	1	10

ASSETS.

	£	s.	d.
Freehold and Leasehold Property (As per Balance-sheet of 31st December, 1897).	51,150	0	0
Fixtures, Fittings, and Furniture £1355 19 8			
Less 5 per cent. written off for depreciation . 67 16 0			
	1288	3	8
Engravings . . . (As per Balance-sheet of 31st December, 1897.)	555	0	0
Contents of Library as on December 31st, 1897	7941	0	0
Added in 1898 . £260 13 2			
Less Depreciation (50 per cent.) £130 6 7			
	130	6	7
Stock of "Climates and Baths".	8071	6	7
Investment—"Permanent Endowment Fund"	142	1	9
(New South Wales 4 per Cent. Inscribed Stock).	326	7	3
Sundry Debtors for Rents .	240	18	3
Cash at Bank and in hand .	336	4	4
	£62,110	1	10

NOTE.—The Society is also possessed of £672 5s. 9d. Consols, and £1 0s. 7d. in cash, but as the sum in question is held in trust for a specific purpose, viz. the Marshall Hall Memorial Prize Fund, the capital sum has not been included amongst the assets of the Society.

Audited and found correct,

TOM MINDY

LIST OF PAPERS.

N.B.—The Council of the Royal Medical and Chirurgical Society deem it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, and opinions set forth in the various papers which, on grounds of general merit, are thought worthy of being published in its *Transactions.*

LIST OF PLATES.

ADDRESS

OF

THOMAS BRYANT, F.R.C.S.

PRESIDENT,

AT THE

ANNUAL MEETING, MARCH 1st, 1899.

GENTLEMEN,—The great honour you conferred upon me last year by electing me your President has been enhanced by my re-nomination upon the present occasion, and for this distinction allow me to thank you as deeply as I can. The work of the Society has been fairly smooth, and for this I am indebted to the kindness and courtesy of every member of the Council, as well as to the consideration and business capacities of our able Secretaries ; one of whom, I regret to say, we are about to lose—for Dr. Norman Moore has used his high talents to the great advantage of this Society, and to its harmonious working, for which I personally and in the name of the Society warmly thank him.

I may certainly say that the work of this Society during the past year has been satisfactory. The addition

to the list of Fellows has been thirty-five, and the loss
fourteen; the papers and discussions upon them have
been well up to the average. I should, however, have
been more satisfied if the attendance of the Fellows at our
meetings had been larger.

The reports which have been read all tell of good
work, and particularly that of the Treasurers, for they
have wisely introduced under the guidance of a skilled
auditor, Mr. Mundy, a new and clear system of accounts,
by which the Fellows of the Society will not only find
recorded the receipts and payments of the Society during
the past year, but a complete revenue account showing
the exact financial position of the Society on December
31st, 1898. The Treasurers assure us also that there
will be no difficulty in the future in continuing to keep
the accounts in their present form. For this good service
I am sure the Fellows of the Society will express their
warmest thanks to our Treasurers.

That I have no untoward events to record is a matter
of congratulation, and I am pleased to add that as my
remaining duty I have to bring before your notice the
biographical records of but eleven Fellows.

DEATH-ROLL.

Date of Election.	Name.	Age.	Date of Death.
1850	Quain, Sir Richard, M.D.	81	March 13th, 1898.
1842	West, Charles, M.D.	81	March 19th, 1898.
1892	Cotterell, Edward, F.R.C.S.	41	April 5th, 1898.
1843	Lee, Henry, F.R.C.S.	81	June 11th, 1898.
1867	Garlike, Thomas, F.R.C.S.	82	July 19th, 1898.
1850	Roper, George, M.D.	74	August 14th, 1898.
1851	Jenner, Sir William, M.D., G.C.B.	83	December 11th, 1898.
1856	Hare, Charles J., M.D.	81	December 15th, 1898.
1893	Kanthack, Alfredo Antunes, M.D.	35	December 21st, 1898.
1871	Rutherford, William, M.D.	60	February 20th, 1899.
1888	Arkle, Charles, M.D.	37	February 22nd, 1899.

Sir Richard Quain died at his residence in Harley Street
on March 13th, 1898, in his eighty-second year, after a life
full of activities, and a long and wearisome illness, which
never seemed to have dulled his active intelligence, or his

interest in the work of the General Medical Council, of which he was President when he died.

He became a Fellow of this Society in 1850, and later on, a member of its Council and a Vice-president, but apparently he never took any active part in its proceedings.

He was born at Mallow, near Cork, in 1816, and was the eldest of eight children. He was educated at the Diocesan School of Cloyne, at the same time as his cousin, John Richard, afterwards Mr. Justice Quain, and Edward Sullivan, subsequently Lord Chancellor of Ireland.

When he left school he was articled to a medical practitioner at Limerick, and in 1837 came to London and entered as a medical student at University College, where one of his cousins, Jones Quain, was a teacher of anatomy, and Richard Quain, another cousin, was a surgeon. At this school young Quain soon became conspicuous for his industry and intelligence, which gained him many class prizes, and in due course he was appointed House Surgeon, and later on House Physician to University College Hospital.

The latter position he held for five years, and during this time he became a graduate in the University of London, and in 1842 took the degree of M.D.

In 1846 he became a Member of the College of Physicians, London, and in 1857 was elected Fellow. In 1872 he was Lumleian Lecturer, and in 1885 Harveian Orator, but he never reached the position of President.

In 1848 Dr. Quain received his first hospital appointment as Assistant Physician to the Brompton Hospital for Diseases of the Chest; in 1855 he became Physician, and it was when working in this institution that he obtained the reputation which gave him a place in his profession and led to fame. He retired from the active staff in 1875.

In 1869 he was elected President of the Pathological Society, having been one of its original members, and in 1852 one of its secretaries. Many Fellows here present

will remember the humorous speech he made at its Jubilee Meeting in 1896.

From early in life Quain took an active part in the public work of his profession. Thus in 1860, when forty-four years of age, he was selected by the Queen in Council to be a member of the Senate of the University of London, and every one knows how well he justified the choice.

In later years he became, as a member of the Senate, Chairman of the Brown Institution; indeed, it has been said that he was mainly instrumental in procuring the acceptance of the Brown Trust by the University of London; and if so, the gratitude of the profession is indeed due to his memory, for from the Brown Trust the profession has already derived much benefit, and in the future we may fairly look for like good results.

In 1863 he became a Crown member of the General Medical Council, which had then been established five years; and in the year following he was elected its treasurer. In this capacity he took an active part in all the work of the Council, so that in 1891, at the death of Mr. John Marshall, he became its President, and as its President he died, after having served the Council for thirty-five years.

With what care, thought, intelligence, and skill he managed the general work of the Council; and the genial way in which he at the same time helped its heterogeneous human elements to do their work in a spirit of harmony, which was rarely broken, can never be forgotten by those who have been subjected to his influence; although at times, it must be admitted, he allowed his own personal views and objects to predominate over those which were entertained by the Council generally.

That he did so under the conviction that he was in the right, and others were in the wrong, may fairly be assumed; but none of us can admit that this line of conduct was one which should be imitated, for in Council business, Council decisions should always be binding, and

the presidential privileges and influence should only be used outside the Council chamber to promote Council views.

Still Sir Richard Quain did much for the General Medical Council, and for the public through its actions. His attention to his duties was thorough, and I would point out that as a member of the Pharmacopœia Committee since 1864, and as its Chairman since 1874, he took a very prominent part in the preparation of the first two issues of the work; and with respect to the one last published, the proofs and revised sheets of the whole work passed through his hands. Indeed, the interest he took in the work was so great, that it is a common subject of belief that it was due to Sir R. Quain's strong desire to see the work completed that he failed to send in his resignation of his position of President as soon as he found himself incapable of discharging its duties.

He, moreover, used his personal influence with admirable effect in finding house accommodation for the Council in its time of need; for it is not to be disputed that it was through his exertions in 1874 that a lease was originally obtained from the Government of the premises then known as the Royal College of Chemistry, but now occupied by the Council; and it is equally sure that it was through his personal efforts, prolonged through the years 1895 and 1896, that the Council were able to purchase the valuable freehold of their premises, and to erect a council-chamber which of its kind is all that could be wished.

With respect to Quain's position as a physician and practitioner, there can be no question as to its having been a success; such success came to him early in life, and was continued up to a very short period before his death. Doubtless much of this was due more to his personal qualities and active intelligence than to high professional qualifications, for his capacity of making and keeping friends was even early in life very striking, and

as he increased in years the value of these attributes was not lessened.

It is not in my power to say that he contributed much to the science of his profession. His only valuable and original contribution to pathology formed his first and last communication to this Society in 1850, " On Fatty Degeneration of the Heart ; " and the knowledge imparted by that communication is now part and parcel of our common knowledge.

But I must record a somewhat pathetic incident associated with his literary work. In June, 1897, though sick and suffering from the effects of an abdominal operation rendered necessary by an organic disease, Sir R. Quain quitted the Presidential Chair of the General Medical Council for a brief period during its session, in order that he might read before the Royal Society, of which he had been a Fellow since 1871, a paper on " The Mechanism by which the First Sound of the Heart is produced."

How far his theory as then propounded has been accepted I know not, but such pluck and energy as Sir R. Quain displayed on that sad occasion certainly claims our sympathy and respectful admiration.

In 1882 a Dictionary of Medicine was published under his name, as editor, and it contained several articles from his pen. The book, I believe, has been a great success, and the editor's work upon it was not light.

For an estimate of his work as assistant and full physician to the Brompton Hospital I have received kind help from a valued Irish friend and learned physician who was Sir Richard Quain's colleague.

He writes that Quain had the art of rapidly extracting desired information from his ignorant patients, and that his rapidity and accuracy in diagnosis were very striking. If he did not enter much into the scientific aspect of disease, and failed to add anything to our knowledge of the pathology of tubercle, he was ever shrewd and practical in treatment. But it was in committee work that Quain chiefly displayed the peculiarities of character

which afterwards led to his success in life, for in debates
on difficult questions, his readiness of reply, tact, and
ability were always effective.

After a long intercourse with him, my friend adds, " I
conceive that he owed his rise in life to a personal power
of pleasing, and to a knowledge of those details of life
and character by which man masters men. He possessed
the Irish character in perfection, or rather in exaggera-
tion,—great shrewdness, a rapid appreciation of facts, a
good memory for faces and details, with an easy manner,
often approaching joviality, free and easy to all, without
regard to rank. These, and a capacity for seeing the
humorous in every scene—even the saddest—of human
life, enabled him to captivate every stranger who ap-
proached him. His *bonhommie* attracted men of all ranks
and professions. With authors, actors, and artists he
was an especial favourite, and as an after-dinner com-
panion he was unrivalled."

Six days after the death of Sir Richard Quain, and at
the same age, eighty-one, a very distinguished member of
this Society, and a Past-president, *Dr. Charles West,* peace-
fully passed away. His life had been a busy and pro-
ductive one, and as founder of the Children's Hospital
in Great Ormond Street, Lecturer on Midwifery at the
Middlesex and St. Bartholomew's Hospitals, and author of
two such works as those ' On the Diseases of Infancy and
Childhood ' and ' On the Diseases of Women,' he is not
likely to be forgotten by future generations

He was born in London in 1816, and was the son of a
Baptist lay preacher, who, having had a " call " into
Buckinghamshire, started after a few years a boys'
school, in which the son received his early education.
Subsequently he went to a school at Totteridge kept by
a Mr. Wood, whose son, as Dr. Wood, was afterwards the
well-known Chairman of Convocation of the University
of London. He was apprenticed at the age of fifteen
to a Mr. Gray of Amersham, a medical man in his father's

village, and there learnt, as he himself admitted, the value of compounding medicines, and the pleasures and advantages of a familiar acquaintance with Shakespeare, which probably fostered the literary taste and graceful style of teaching which were so conspicuous in all Dr. West's later work. Indeed, all who remember the calm, deliberate way in which West spoke, the clearness of his language, and the care with which every sentence was expressed, will endorse the truth of what has been stated, for West would never allow himself to be accused of carelessness in his speech or writings, or to express his thoughts in other language than the best.

After a two years' residence at Amersham he entered as a student at St. Bartholomew's Hospital, and during the two years he worked there he became distinguished for his industry and ability. He wished to go to Oxford after leaving St. Bartholomew's, but his father's theological opinions were against this; so he went to Germany, and during the year he studied at Bonn he gained the University Prize for a Latin Essay on "The Female Pelvis and its Influence on Parturition;" and this success was probably the starting-point of the line of professional life he subsequently followed. When he left Bonn he went to Paris, and on to Berlin; at both places he spent a year, and at Berlin in 1837 he took his doctor's degree.

He then returned to London and bought a partnership in a City practice, which does not seem to have been a success, for he had evidently much leisure, which he spent well in working for some months in the wards of his old school, under Dr. Latham, after which he went to Dublin, where he became an indoor pupil at the well-known Rotunda Lying-in Hospital, under the late Dr. Evory Kennedy, and at the same time he followed the teaching of such distinguished men as Graves and Stokes. He returned to London in 1839, became clinical clerk to Sir George Burrows, published a translation of Naegele's work on 'Obstetric Auscultation,' and at the Infirmary

for Children in the Waterloo Road he began the study of the diseases of children, upon which he subsequently became such a distinguished authority. In 1842 he was appointed Physician to that institution.

From this time he worked steadily at midwifery and the diseases of women and children, and it has been recorded that in the early years of his work, and " in the hope of some day having the opportunity of lecturing, he prepared a complete course of lectures on midwifery." In this hope he was not disappointed, for in 1845 he was appointed Lecturer on Midwifery at the Middlesex Hospital, and in 1848 lecturer on the same subject at his old school of St. Bartholomew's, a post he well filled for twelve years.

During his connection with the Middlesex Hospital he gave a course of lectures on the diseases of children, which were published as a book in 1848, and during his connection with St. Bartholomew's Hospital he published his work on 'Diseases of Women.' Both works have passed through many editions in many languages, and the sound practical sense which they contain, and the graceful language in which such full information was presented, have caused these works to be highly appreciated by all professional workers.

The story of West's efforts to convert the Waterloo Road Children's Infirmary into a hospital, with his failure and its causes, would doubtless be an interesting chapter to read, as would also a full account of his establishment of the now well-known and appreciated Hospital for Children in Great Ormond Street in 1852, with his introduction in 1862 of the trained and educated gentlewoman as nurse into its wards.

What has been made public upon these matters has only excited an appetite to know more. In this place I can only recognise the value of the work that was done and give its doer full credit, for it was good work, which has proved abundantly fruitful in many ways. It has been recorded that up to the very end of his life his

anxiety for the welfare of the Children's Hospital was very great.

To give anything like an account of West's literary activity would occupy more of your attention than I can claim. It was unceasing during his whole career.

An essay containing an account of an epidemic of typhus fever as seen in cases admitted into St. Bartholomew's Hospital in 1837–8, which was published in the ' Edinburgh Medical and Surgical Journal' for April, 1838, was his first paper.

In 1840 he published a translation of Müller's work on the ' Structure of Cancer and other Morbid Growths.'

He wrote for the ' Penny Cyclopædia,' ' Biographical Dictionary,' and the old and valuable but now extinct ' Medico-Chirurgical Journal' for many years.

In the middle period of his life, besides the grand books already mentioned, he in 1874 gave the Harveian Oration, and published a book on ' Harvey and his Times.'

In 1852 he delivered the Croonian Lectures at the Royal College of Physicians on ulcerations of the os uteri, and undoubtedly did much good in checking practices which seemed to be verging on abuse. In 1871 he gave the Lumleian Lectures.

Within the last few years, in 1887, he brought out a new edition of his ' Mother's Manual of Children's Diseases,' and so late as 1896 he published a valuable work on ' The Profession of Medicine : its study and practice, its duties and rewards,' this book being an enlargement of his address at the opening of the St. Bartholomew's Medical Session in 1850.

As a practitioner of medicine West was undoubtedly successful. With children his influence was always soothing, and the skilful sympathy he displayed so satisfactorily with the little ones was not less effective with their mothers. Indeed, it has been said his consulting room was as full of those who sought his advice, when in mental sorrow or distress, as of others who wanted his

professional aid in their bodily ailments, which he understood so well how to treat.

. Physically, Dr. West was not a strong man, and on that account he gave up his London work in 1880 and went to Nice, where he practised for five years, and then returned to London and to work, although he never really cared or sought for private practice. He was somewhat disappointed, however, that he was unable to find an opening in one or other of the London children's hospitals, where he could follow up the work his heart yearned for—the clinical study of children's diseases.

In 1891, after a severe attack of neuralgia, which told much upon his strength, he took to travelling, although much of his time was spent in the study of history and literature; and as he could read in six languages, his field of work was very wide. Indeed, his intellectual powers and ability to work were of the highest order, and they were maintained up to a few days before his death.

He died in Paris, where as a Foreign Fellow of the Paris Academy of Medicine he was well known and honoured, and by his own request was buried at Chislehurst. He married twice, and his second wife is still living.

Thus in outline have I laid before you the main facts of the life of a physician and former President of this Society, who in his day played many parts and played them well. As a worker he was unceasing, as a thinker close, as a searcher after truth strenuous, as a writer and lecturer almost perfect, for he possessed a knowledge of the English language which was that of a master. That he had imperfections goes without saying; but it is neither my province nor pleasure to seek them out; for his good qualities are those we and those who follow us ought to imitate.

Edward Cotterell, who died on April 5th, at the age of forty-one, from an attack of pneumonia following influenza, was a man of promise, and there can be little doubt that

he would have taken a good position amongst the surgeons of his time had his life not thus been shortened. As a student of University College Hospital he had a distinguished career, having there gained the Atkinson-Morley Surgical Scholarship, and was one of Mr. Christopher Heath's house surgeons.

He became a member of the Royal College of Surgeons of England in 1880, and in 1891 took the Fellowship. Between these dates he practised at Bicester, where he made a good local reputation by his surgical work. He came to London in 1891, and was appointed surgeon to out-patients at the Lock Hospital. He favoured the treatment of syphilis by intra-muscular injections of soluble mercurial salts, as ably advocated by Surgeon-Major Lambkin in the ' British Medical Journal' for February 19th, 1898. He was editor of the second edition of Mr. Alfred Cooper's work on syphilis. He took up enthusiastically the treatment of "roarers" by tracheotomy. He published also the 'Pocket Gray,' a *vade-mecum* of anatomy, which reached a fourth edition, and some few other papers.

Mr. Henry Lee, who had been a Fellow of this Society for fifty-five years, one of its Vice-Presidents, and a contributor of fourteen papers to its ' Transactions,' many of which were of great value, died on June 11th, 1898, at the age of eighty-one years. He began his professional career at St. George's Hospital in 1834, and became a Fellow of the Royal College of Surgeons in 1844.

In 1847 he was appointed assistant surgeon to King's College Hospital, which had been then newly established, and also to the Lock Hospital. But in 1861 he returned to his old school of St. George's as one of its assistant surgeons, and in 1863 he became full surgeon. In 1878 he retired from this post and was made consulting surgeon, a position he held up to the time of his death.

Physiology and pathology were his favourite studies in early life, and the basis of all his later work when he

acted as curator of the museum of St. George's Hospital, and later as lecturer on physiology ; indeed, his first paper read before this Society and published in its ' Transactions ' was on the " Deposition of Fibrin in the Lining Membrane of Veins," a paper for which he rightly obtained much credit.

His connection with the Lock Hospital naturally led him to study the broad subject of syphilis, and a paper of his on " Calomel Fumigation in Primary, Secondary, and Tertiary Syphilis," based on his practice at the Lock Hospital, was read before this Society in 1856. This paper was without doubt a valuable one, and his advocacy of a clean and painless method of introducing mercury into the system for the cure of constitutional syphilis must ever be regarded by his professional brethren with respectful gratitude.

His Hunterian Lectures on syphilis in 1874, and his article on syphilis published in ' Holmes's Surgery,' are also worthy of close study.

In all the good work Lee did he was careful and painstaking—in his operations he was so particularly—and consequently he was considered to be successful. As a consultant he was very trustworthy, and as a teacher was much sought after, for his views were always given thoughtfully and clearly, and to the senior students in a kindly way. Indeed his kindness of heart and genial ways endeared him to all who came into friendly contact with him. His appearance at any medical dinner was always hailed with pleasure, and his old colleagues on the Council of the Royal College of Surgeons, with whom he had served for his full term of office from 1870, always made much of him when he subsequently attended any of the dinners of the College Council Club. Indeed I can fully endorse the conclusion of a friendly biographer that " He will be long remembered by his many friends, pupils, and colleagues as a man who used considerable talents to worthy ends—a good surgeon, a good colleague, and a good friend."

George Roper, who died on August 14th, 1898, the day before his seventy-fifth birthday, was one of an old and much respected Norfolk family. He received his medical education at Guy's Hospital, and passed his professional examinations in 1847. In 1849 he established himself in general practice in the East End, where he was successful to an extreme degree, his kind nature much enjoying the friendly confidences of his poorer patients. In 1866 he retired from this practice, in which he had gained not only a great liking for, but great experience in, the practice of midwifery; and as he had made up his mind to follow this branch of practice as a speciality, he went to school again and entered the University of Aberdeen, where he attended his classes with the youngest. He worked so well and steadily that in 1873 he took his M.D. degree at Aberdeen, and a few months later became, in 1874, a Member of the Royal College of Physicians of London. Not long after these successes he became attached to the Infirmary for Children and Women in the Waterloo Road, and also Physician to the Royal Maternity Society. He joined also many of the working medical societies. He had been a Fellow of this Society since 1850, and in 1879 he served as a member of its Council.

Dr. Roper, who was well known to me, was a man of wide culture, sound judgment, and deep sympathies. He had likewise a charming manner, which won the hearts of all his friends. He had a full knowledge of human nature, which was helped by much travel—consequently his society was greatly valued. When he retired from professional work in 1889 to live in Norfolk—his native county—he was much missed, and during the nine years he spent at his country home he was everybody's friend. Dr. Roper was, indeed, a typical practitioner of medicine, and it would be well if more of his kind and calibre were to be found in our profession.

The late *Thomas W. Garlike*, F.R.C.S., who died on

July 19th, 1898, aged eighty-two years, was apprenticed to Messrs. Flower and Leach, of Chilcompton, Somerset, from whence he came to London and entered at St. Bartholomew's Hospital.

In 1841 he went into practice at Rickmansworth, near Watford, Herts, where he was for eighteen years. He was medical officer to the Watford Union, and had a very large practice, including many adjoining parishes. He performed successfully many surgical operations ; one, a case of successful amputation of the thigh for senile gangrene in a patient eighty years of age, was read before this Society in 1851. He also practised for many years at Tulse Hill, Brixton, and in 1877 he retired into private life at Ealing Dean.

By the death on December 11th, 1898, at the ripe age of eighty-three, of *Sir William Jenner*, G.C.B., Baronet, F.R.S., Her Majesty's physician and friend for upwards of thirty years, and a past President of the Royal College of Physicians for seven years, the profession was deprived of a personality which was greatly prized, and the public of a friend who was highly valued. In this Society he had been a Fellow from 1851, had communicated two papers, and served as Vice-president.

By his industry and ability all through his professional career Jenner had steadily but surely mounted the ladder of fame. In his early years he had had to rely upon his best friend—his own exertions ; in his later years, which were the fruits of his earlier, with some favourable circumstances he advanced rapidly, but I am disposed to think that this steady professional success was due principally to the fact that from his student days he had fully recognised the equal value of the two fields of knowledge upon which the science of medicine and surgery is to be acquired or advanced, namely, the clinical and the pathological. For the wards and the post-mortem room were without doubt his favourite places for professional work, and consequently he found, as the best of our physicians and surgeons have

ever found, that the combined study of these twin fields of observation leads to the happiest results.

As an observer, Jenner's keenness was of the Hunterian type, for nothing escaped his scrutiny. As a reasoner he was singularly clear and logical, for his reasoning was ever based upon the facts before him, and he had little faith in theories or in opinions which were not founded on such data. I well remember, when he was President of the Pathological Society in 1873, some member of the Society exhibited a specimen of a suggestive character, and the President asked the members for any information they might possess concerning like or allied specimens, but added, " I do not 'ask for speculative theories or opinions unsupported by facts."

As a teacher and lecturer Jenner was said to be perfect, and to hear him describe a case with its clinical and pathological features presented in perfect order, was a lesson not to be forgotten. It was not my privilege to have enjoyed any of his clinical teaching otherwise than as a practitioner, but with a mind so clear and a knowledge so full it can readily be understood that his clear words came naturally from his clear thoughts ; for is it not true that confused speech is, as a rule, the result of confused thoughts, and that when clear thought exists clear teaching follows ?

And here I may be excused if I quote a few of his recorded sayings, which I believe to be too good to be passed over.

Dr. G. V. Poore relates that in the opening lecture of a course of systematic medicine, and when unrolling his manuscript, he began, " The great aim of the physician is to prevent disease ; failing that, to cure ; failing that, to alleviate suffering and prolong life."

To a student he would often say, " Why do I ask you questions ? " and if in response the student replied, " I suppose to find out what I know," Jenner would immediately answer, " Not at all ; it is to find out what you do

not know, in order that I, by my teaching, may lessen your ignorance."

In his teaching, which was undoubtedly dogmatic, he would often say, " That is the opinion I have come to from my observation ; " but would add to the students, " You must *test* it for yourselves by your own observations."

" In typhoid fever, when delirium begins, headache stops ; headache and delirium always mean meningitis."

" When in doubt about alcohol in typhoid, don't give it ; but when in doubt in typhus, give it."

" When you see a child suffering from obstinate vomiting and constipation, always think of meningitis."

In writing a lecture he advised a favourite pupil, " First state your general conclusions, then give your cases, and your reader will be able to carry along with him the clue which you have previously given him, and see how far you establish your contentions."

At a meeting held to promote a memorial to his great friend Dr. Parkes, he spoke of the " D " trap, and said, " I call it the double ' D ' trap." A certain thrill ran through his audience, who wondered what was coming, until he added after a dramatic pause, " because it deals out death and disseminates disease."

I must, however, add one more of his good sayings, which I believe to have been his best. It is one which I as a teacher have always regarded as being most important—" More mistakes are made by want of looking than by want of knowing."

Jenner's written contributions to our science well illustrate what I have said. They have not been voluminous, but they have been full of facts, logical deductions from facts, and are original. His paper " On the Identity or Non-identity of Typhoid and Typhus Fevers," which was read before he had joined our Society and published in our ' Transactions ' for 1850, is one which fairly marks an era in modern medical days, and this was doubtless the outcome of his study, commenced in

1847, of the cases of continued fever admitted into the London Fever Hospital; for during 1847 and 1848 he made clinical and pathological notes of nearly 1000 cases of acute disease which had been under the care of Dr. Tweedy, the Senior Physician to the Hospital.

In April, 1849, he published in the 'Monthly Journal of Medical Science' an essay upon "The Symptoms and Appearances found after Death in Sixty-six Fatal Cases of Continued Fever observed from January, 1847, to February, 1849," and it was this essay expanded which was read at this Society, and subsequently published as an independent volume. This paper, containing as it did both clinical and pathological evidence of the non-identity of typhoid and typhus fevers, greatly helped the acceptance of the view which was "in the air," that these two fevers, which had been counted as one, were distinct; and since its publication it may be asserted that this conclusion has never been disputed, although it must, in the interest of truth, be stated that by the works of a former Fellow of this Society, Dr. A. P. Stewart, in this country, and of two or three other physicians at home and abroad, evidence had been collected previously suggesting that the two fevers were not alike, Dr. Stewart's paper having been read before the Parisian Medical Society on April 16th and 23rd, 1840, and reprinted by the New Sydenham Society, 1884. In fact, with these two papers before them, although published at an interval of ten years, all workers in the profession, silent or otherwise, have fully confirmed Stewart's and Jenner's general conclusions, which are now accepted and taught as the true views. In two volumes published in 1893 and 1895 Jenner's papers on rickets, emphysema, and diphtheria will be found, with others which are now well worthy of careful study. "They deserve," as Sir William Gowers has written, "reading and re-reading, and must always have a value for those who value logical thought and lucid presentation of the facts that cannot die."

Jenner's success was, however, due to other qualities

besides those I have placed before you, and those qualities were found in the man's personality. His singular kindness of heart, as indicated by his consideration for the sick in every grade of life, was always conspicuous, although in every-day life he at times may have displayed a curtness of manner which might not have given support to this view. Still, in the presence of illness all this was absent, and his gentle voice and quiet demeanour in a sick chamber invariably displayed the kindness of heart which lay behind. All his friends will recognise the truth of these remarks; and to support them have we not been told by our gracious Queen that he was to her not only a most able physician, but a true and devoted friend? and for this position such a thing as a kind heart must have been an essential. His uprightness of character and sterling honesty of purpose were likewise very striking. The slightest tinge of anything like self-advertisement or quackery was offensive to him, and in his own life was entirely absent; and the careful way in which he kept from public gossip all and every secret with which his public life must have made him acquainted was worthy of all respect and imitation.

Sir William Jenner in counsel was very strong, and upon every question to which he had applied his mind, he held decided opinions. He also enforced them with no lack of vigour. All who ever met him in conference, whether at the College of Physicians as President, the University College as member of the Senate, and in the council-chambers of the many societies of which he had been president, must have fully felt the weight of his personality; and if his opposition to a suggested line of action, in one or two instances may be regretted, upon the whole his influence was generally for good. It must, however, be admitted that Jenner had certainly a weak point in his intolerance of opposition, and I think it may fairly be said that as an administrator he was less fitted to be the head of a constitutional than of an autocratic government.

I do not propose to take up your time by enumerating the different professional positions he held and the different honours and distinctions he acquired as he gradually mounted the ladder of fame since starting as a student at University College Hospital.

Every new position he acquired and every distinction he won was worthily gained, each success in his life seemed naturally to lead to a higher, and so on to the highest; and when, from illness, he had to retire from all professional work, he took with him into his retirement the friendship of his Queen and the Royal Family, and the respect and admiration of the profession he had so long adorned. And now he has gone to his long resting-place, at the great age of eighty-three, we can truly say that he was well worthy of the distinctions he had gained, that he had done his work in life in a manner to be admired and followed; and that, whilst we mourn his loss, we may gain strength from the conviction that whilst God buries His workmen, He still carries on His work.

By the death of *Dr. Charles J. Hare,* in his eighty-first year, on December 15th, 1898, the Society has lost a warm friend, for he joined the Society in 1856, had been one of its Vice-Presidents in 1894-5, and from 1887 to 1894, a period of seven years, served as Treasurer, this lengthened period of treasurership having included the duties of a member of the Building Committee from 1889 to 1892, by the labours of which we are now housed in our present splendid quarters, and become the owners of a fine property. For the work Dr. Hare with others did for us during that period of our Society's history we must for ever be deeply grateful. We must not forget also that Dr. Hare rendered other services to this Society by representing us at the International Medical Congress at Philadelphia in 1876, and at Rome in 1894, as well as at the Centenary of the Société de Médecine of Paris in 1896.

Charles J. Hare was the son of the late Mr. Samuel Hare,

F.R.C.S., of Leeds. He received his medical education at Cambridge; University College, London, and at Paris; and he took his degree of M.D. at Cambridge in 1847, when twenty-nine years of age.

In 1850 he was appointed Assistant Physician to the University College Hospital on the same day as was his lifelong friend Sir William Jenner, and he survived his old friend only four days—Jenner having died on December 11th, and Hare on December 15th, 1898, both men being members of the Consulting Staff of the University College Hospital when they died.

In 1862 Hare became full Physician to the University College Hospital, in the place of Dr., now Sir A. Garrod, who had resigned; but he gave up this position in 1867, having damaged his sight by microscopic work : during these five years he had, however, done good work in the School as Professor of Clinical Medicine.

As a professional writer Hare never did much, and beyond some contributions to the medical journals of the day, and a reprint of an interesting address he gave in July, 1883, as President of the Metropolitan Counties Branch of the British Medical Association, on " Good Remedies out of Fashion," I have nothing to record.

His capacity for letter-writing was, however, very great, and his letters were always good ; in all his work he was methodical, exact, and conscientious.

As a companion he was ever acceptable, for he loved talk, and never failed to give pleasant reminiscences of his own and others' lives, without a single taint of jealousy or ill-humour. His manner was always cheerful and courtly, and his aspect fresh and that of health. His courage was always good, and even when he believed that he was the subject of carcinoma of his œsophagus this courage did not leave him.

When on his death-bed his last coherent message, taken down by Mr. MacAlister as follows, was sent to this Society :

" Tell the Council the Society has my unfailing love

and respect, and I pray it may enjoy always honour and prosperity. . . . . prosperity."

And to the profession he so well adorned we may accept as to ourselves the following New Year card which he sent to a friend, and which one of his biographers so aptly writes was at once the expression and the secret of his bright life :

"1898.—Take thy New Year with thankfulness ; but is it not well, starting afresh with hope and trust, to commence a new year every morning, determining to leave, as far as may be, all worries (which 'have killed their thousands') and all troubles and vexations to the last year's account —closed the night before ?"

By the premature death, at the age of thirty-five, of *Alfredo Antunes Kanthack*, M.D., and B.Sc.Lond., F.R.C.S.Eng., F.R.C.P.Lond., and Fellow of this Society since 1893, the world of science has lost a worker of wonderful ability, for as a teacher of pathology at his London School, St. Bartholomew's Hospital, and afterwards at the University of Cambridge, he made a name and reputation of which a man twice his age might have been justly proud.

He was born in Brazil in 1863, where his father was Her Majesty's Consul. He was educated from the age of eight to eighteen at Hamburg and Luneburg, and up to his fifteenth year never displayed much power. In 1881 he came to England, and when a student at University College, Liverpool, took consecutively, after his matriculation, the B.A., M.B., B.Sc. of the London University, and all with honours.

In 1887 he came to London, and whilst at St. Bartholomew's Hospital became a Member and Fellow of the Royal College of Surgeons.

In 1889 he went to Berlin, and there, whilst working under Virchow and others, he made a name which is not likely to be forgotten.

In 1891 he became John Lucas Walker's student at Cambridge, and thus resumed his research work.

In 1892 he left Cambridge, and returned to Liverpool to start in practice ; but this was not to be, as he was appointed to the post of Medical Tutor at the Royal Infirmary, and Demonstrator of Bacteriology. From this position he was, however, in 1893 attracted back to St. Bartholomew's as Lecturer on Pathology, and in 1897 he was drawn away even from London to Cambridge to take the place of Professor Roy, who had died, and to become Professor of Pathology in the University of Cambridge. In that position he died of some malignant abdominal growth after a few months' illness. During all these years he worked unceasingly.

In the year 1890 he was appointed by the Royal College of Surgeons of England one of the Commissioners with the late Dr. B. Rake and Dr. Buckmaster to investigate the question of leprosy in India, under the direction of the National Leprosy Fund; and splendid work was there done, as indicated by the Commissioners' Report on Leprosy.

In 1889, when at Berlin, he published a paper on " The Histology of the Larynx " in ' Virchow's Archiv,' which established his reputation as an original observer.

On his return from India he published papers which were deemed worthy of a place in the ' Proceedings of the Royal Society.' He demonstrated the parasitic nature of the Madura disease, and its close resemblance to actinomycosis. In 1898 he published a most interesting report on the inquiry which he had been conducting, with the aid of Mr. Durham and Mr. Blandford, into the tsetse fly disease.

Besides these papers, he published many others in the scientific journals, and likewise ' A Manual of Practical Morbid Anatomy ' in 1894, and ' A Handbook of Practical Bacteriology ' in 1895.

In the same year he also won the Jacksonian Prize Essay, given by the Royal College of Surgeons of England, on " Tetanus."

In general literature Kanthack was widely read, and having seen much of the world, held no narrow views, and was a good linguist. His industry was untiring.

As a companion he was all that could be wished, for with his full knowledge and powers of exposition, set off by a natural charm of manner, he had everything to make him popular.

Every one must lament his loss, and re-echo Prof. Virchow's hope when he heard of Kanthack's death— "May English Medicine never lack such men."

On February 22nd the death of *Dr. William Rutherford*, of Edinburgh, a distinguished non-resident Fellow of this Society, has to be recorded. He died from influenza at the age of sixty. He became a Fellow of this Society in 1871, and is described in our list of Fellows as Professor of the Institutes of Medicine in the University of Edinburgh. He passed through his student's career at Edinburgh University with marked success, and taught anatomy at Surgeons' Hall under the late Sir John Struthers. He then studied at Berlin, Vienna, and Paris, and returned to his former school to be appointed University Assistant to the late Professor John Hughes Bennett in 1865.

In 1869, when only thirty years of age, he came to London to hold the appointment of Professor of Physiology in King's College. He occupied this position for five years, and for three of these he was likewise Fullerian Professor of Physiology in the Royal Institution.

In 1874, when Professor Hughes Bennett resigned his post as Professor of Physiology in Edinburgh University, Dr. Rutherford was appointed in his place, and there he laboured till the close of his life, having considerably advanced the branch of knowledge with which he was so well acquainted, and helped by his industry, teaching, and example, to mould the minds of countless students of his University to pursue right methods of research, and to apply their knowledge to practical purposes.

He was much admired by his pupils and colleagues, and to his University his loss must be very great.

Charles Joseph Arkle, the fifth son of the late Benjamin Arkle, Esq., was born in 1861 at Liverpool, and received there his early education. Commencing his medical studies at University College, London, in 1879, he soon made his mark among the men of his year, distinguishing himself more particularly in his clinical and practical work, and obtaining the much coveted Fellowes Medal for Clinical Medicine, and the Atkinson-Morley Scholarship for distinction in surgery. He graduated as a Bachelor of Medicine of the University of London in 1885, and proceeded to the Doctorate in 1887. In 1888 he was elected a Member of the Royal College of Physicians, and only last year was admitted to the Fellowship. In 1888 he became a Fellow of this Society.

In University College Hospital Dr. Arkle held all the resident appointments, being House Physician to the late Dr. Wilson Fox, and House Surgeon to the late Mr. Marcus Beck. By both his masters he was highly esteemed, and in 1887 was in constant attendance upon Dr. Fox at the time of his death. In 1889 he was elected Resident Medical Officer to the Hospital, and held the post for two years.

Having thus obtained a very sound training in clinical work, Dr. Arkle spent more than a year on the Continent, studying chiefly pathology in Berlin, Vienna, and Paris. Soon after his return he was appointed Medical Registrar to the Hospital for Sick Children, Great Ormond Street.

In 1892 he began his connection with Charing Cross Hospital as Pathologist and Curator of the Museum. In 1895 he was made Assistant Physician to the Hospital and Medical Tutor in the School, where for several years he was also Teacher of Bacteriology. For two years he had been Vice-Dean of the Medical School, his term of office having by a strange coincidence come to an end on the very day of his death. Since 1897 Dr. Arkle had

also held the appointment of Assistant Physician to the Hospital for Consumption, Brompton. As a teacher he had already gained considerable success, and with his pupils he was a universal favourite.

Charles Arkle was a man of wide and varied knowledge and many interests. In his student days his favourite game was football, in which he excelled so greatly that in 1887 he was chosen to form one of the team to represent the South of England against the North. He was also an experienced mountaineer, and for many years had spent his summer holidays in Switzerland with his eldest brother, both being prominent members of the Alpine Club.

As a man, Charles Arkle was a fine example of an open-hearted north countryman. It was his scrupulous fairness, his kindness, and his keen sense of humour which made him a favourite with his patients and those with whom he worked, and so much beloved by his many friends, both old and young. In the conscientious way in which he discharged his duties and cared for the welfare of his patients he clearly showed the influence of his old master, Marcus Beck, to whom many a University College man owes so much.

Dr. Arkle died, after an illness of only a few days, of acute pneumonia, on the 22nd of February of this year.

FIBRINOUS OR MEMBRANOUS RHINITIS

AND ITS

RELATION TO DIPHTHERIA

BY

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(COMMUNICATED BY DR. ALLAN MACFADYEN)

THIS interesting affection has attracted attention in recent years, especially in Germany and America; but, although it is by no means uncommon, little has been written on it in this country. The older observers were of the opinion that the affection was entirely distinct from diphtheria, whilst recent observers, relying chiefly on the results of bacteriological examination, have asserted the identity of the two diseases.

During the past fifteen months (ending October, 1897) I have examined clinically and bacteriologically thirty-six cases of this affection, and have especially endeavoured to trace its connection with diphtheria. This investigation seemed to me important, as it might possibly throw light

on some of the bacteriological problems connected with diphtheria, and also discover a possible overlooked source of diphtheritic infection. The disease seems to be very common. Sixteen cases occurred at the Children's Hospital, Paddington Green, in one year, out of a total of seven hundred new cases,—that is, they formed about 2¼ per cent. of the patients seen. Many other observers have also published series of cases, and, as will appear later, many cases probably do not apply for treatment at all, or if they do, pass unrecognised by well-qualified observers.

Fibrinous rhinitis may be defined as a subacute or chronic affection of the nose, characterised by the formation of a fibrinous, membranous, or shreddy exudation on the nasal mucous membrane.

The affection occurs most commonly in young children, and commences with the ordinary symptoms of catching cold. The nose becomes stopped up, may appear a little red and swollen, and there is a more or less profuse watery discharge. The child may be a little feverish, or complain of headache, seem tired or languid, lose appetite, &c., for the first day or two, but is not sufficiently ill to lie up. As a rule, indeed, it plays about or attends school as usual, and the constitutional disturbance when present at the commencement never persists for more than a day or two. The nasal obstruction and discharge continue, and the latter after a week or so often becomes of a more purulent character. When unilateral such symptoms closely resemble those of a foreign body in the nose, for which the affection is often mistaken. It is for these symptoms, when they have persisted two or three weeks, that relief is usually sought.

On examination there is seen to be complete bilateral or unilateral nasal obstruction, accompanied by a clear watery or muco-purulent discharge, with sometimes more or less irritation of the nostrils and upper lip, or even extensive impetigo.

Fibrinous exudation can be seen over the mucous

membrane of the affected nostril, and sometimes large loose pieces of membrane can be removed with forceps. The general health always seems good, and remains so throughout. The affection lasts on the average four to eight weeks or more, and apparently leaves no sequelæ. Briefly the symptoms are those of a more or less severe cold in the head, and only attract attention from their persistence.

Primary nasal diphtheria has long been recognised, and was well described by Bretonneau. Schuller in 1871, in the case of an infant dying of erysipelas, found the nose lined with membrane, and he is usually considered the first author to have alluded to fibrinous rhinitis. Major in 1886 fully reports a typical case in a girl aged eighteen, in whom the disease lasted three months, but it was not until 1887 that the affection seems to have attracted much attention. In that year Seifert reported three cases. One was an adult and two were children, sisters. Both the latter had also tonsillitis. There was no general disturbance, and he considered the disease distinct from diphtheria.

Moldenhauer reports four cases, and although there was diphtheria in Leipzig at the time, there was no history of direct infection in these cases, and he believes the diseases have no connection.

Also Henoch reports one and Hartmann six cases, all in children, with no spread of infection, although one case occurred in a home for deaf-mutes. In 1888 Ryerson reports a unilateral case, and Hammond one (himself) without suspecting diphtheria ; and Bischofswerder three cases in which he carefully excluded all connection with diphtheria.

In 1889 Gluck and Porter in America express very similar opinions about the disease, stating that it is common and has no connection with diphtheria, but is a severe and chronic form of ordinary coryza.

In 1890 Chapin reports the case of two little children, sisters, and Raulin four cases, three in adults, one of whom

had also a patch of exudation on the pharynx. Both observers exclude all connection with diphtheria.

In 1891 Newcomb reports two cases, Leemans two, Lieven two, Hunt one; and the disease is discussed by others, as MacDonald, Hajek, &c. Up to this date over thirty-three cases have been fully recorded, some observers giving no numbers but stating that the disease is common.

In no case could any connection with diphtheria be discovered, and the disease was by all considered quite distinct. The society to whom Leemans showed his cases declared it was unnecessary to go into the differential diagnosis, no one could confound them.

In 1892 the *rôle* of the Klebs-Löffler bacillus was becoming generally recognised, and the results of the bacteriological examination of these cases were reported and changed the views of its pathology. Baginsky always found the Klebs-Löffler bacillus present, and considered the affection to be a mild form of diphtheria. Park found the bacillus in all of ten cases, in one of which the tonsil was also affected. Stamm and Abbott each found it in three cases. Other observers, however, as Abel, Van Starck, Fränkel, Sedziak, Ritter, &c., failed to obtain it. In this year also Concetti published five cases, in two of which he found the Klebs-Löffler bacillus. His cases should, however, be rather considered as true nasal diphtheria, as he himself calls them. In one of them the disease spread to the larynx, in two cases gave rise to diphtheritic infection, and in one was followed by paralysis of the palate, &c.

In more recent years numerous papers and references to the disease are found, chiefly in German and American literature. Most observers, relying on the results of bacteriological examination, consider the disease a form of diphtheria, and discuss the cause of the mildness of its symptoms.

Perhaps the most careful papers are those of Park and Abbott above alluded to, and those of Ravenel, who reports ten cases, of Gerber and Podack, who observed

six cases, and of Pluder, who also observed six cases. Some results and conclusions of these observers will be discussed later.

The following is a brief analysis of the chief clinical features presented by the thirty-six cases which I have recently observed.

1. *Sex and age.*—Twenty cases occurred in girls and sixteen in boys. Their ages varied from 10 months to 15 years. Nine cases were in children under 4 years of age, twenty-four were aged 4 to 8 years inclusive, one was 9, one 11, and one 15. This proves conclusively that the disease is essentially one of childhood, although cases occurring in adults have occasionally been reported.

2. *Season.*—The cases varied much in frequency with regard to the season of the year; thus of twenty-four cases seen in the last year, from October to March 9 were seen, in April, May, and June only 1, in July 3, in August 6, and in September 5. Thus nearly half the cases were seen in the two months August and September, and looking through my case-book I find a similar result for the previous year.

3. *Nasal obstruction* was constantly observed, the affected nostrils or nostril being completely blocked. This was usually the most prominent symptom, and the one for which relief was sought.

4. *Nasal discharge* was also constantly met with, but varied much in character and amount. In a few cases it was very slight and almost unnoticeable. In other cases a profuse, clear, watery discharge was constantly dripping from the nose. In other cases, again, the discharge was described as a thick yellow matter, or, rarely, as stained brown with blood. Towards the end of the disease " bits of flesh " were occasionally extruded from the nose. In every case in which I have examined the discharge microscopically pus corpuscles were found in it, although, as will appear later, the disease was then late in its course. The discharge was never foetid.

5. *Bleeding* from the nose occurred in about two thirds

of the cases. In most it was slight, and occurred late in the disease, when the membrane was separating, and in many cases was probably induced by traumatism, picking at the nose, efforts at syringing, &c. In a few cases the discharge was frequently blood-stained, and in three there was epistaxis to a very serious amount. Thus bleeding is certainly not a reliable symptom for diagnostic purposes, although by some observers great stress has been laid on it.

6. *Excoriation* of the anterior nares was present in the large majority of cases, although in some it was very slight. In some impetiginous crusts appeared over the upper lip, and in three cases there were pustules on the face and hands. In no case did any membranous deposit occur on these spots, nor could any bacilli be cultivated from them.

7. *Examination of the nose* usually showed a thin, whitish, flaky, somewhat adherent exudation on the inferior turbinals, floor of nose, and septum. The nasal obstruction prevented a view of the deeper parts being obtained. The mucous membrane seemed congested, and bled easily when touched with the probe. It is a striking circumstance that in the great majority of cases the area of fibrinous exudation was absolutely limited by a sharp line corresponding with the anterior extremity of the inferior turbinal; in no case did it extend to the vestibule. In a few cases the fibrinous exudation was limited to a much smaller patch, or a loose piece only of thick membrane was found in the nose.

In eight of the cases, that is nearly one fourth, the affection was strictly unilateral. In these cases the symptoms strikingly resembled those of a foreign body in the nose, and in four of them search was made under an anæsthetic, with the result that large thick pieces of membrane were removed. This membrane resembled in all respects diphtheritic membrane, but usually seemed to be quite loose in the nose.

8. *Examination of the throat* in thirty-two of the cases

yielded a negative result, although in four of these cases there was a history obtained of sore throat at the commencement of the disease. This seemed to be very slight, and lasted but a day or two, and there was nothing apparent when the cases came under observation.

In four cases there were definite lesions in the pharynx. In one case, a boy aged 15, there was present inflammation of one tonsil much resembling follicular tonsillitis. The tonsil, however, was not much swollen nor much reddened, and the exudation from the crypts was more profuse than usual. This exudation was quite soft, creamy material, was easily wiped away, leaving a non-bleeding, slightly congested surface, and spread over the tonsil slightly on to the pillars of the fauces, and on to the lateral pharyngeal band of the same side. The throat was not tender and the glands were not enlarged.

In another case there were one or two white specks on the lateral pharyngeal bands, which were slightly inflamed.

In the other two cases these lateral bands were covered by a thin, whitish, translucent, adherent exudation which extended up to the post-nasal space. There was no swelling or inflammation of the throat, and no pain on deglutition. This condition lasted two or three weeks after coming under observation, and if the history obtained be reliable had previously existed some week or two.

These chronic cases of pseudo-membranous exudation in the pharynx and post-nasal space I have also observed quite apart from fibrinous rhinitis. The affection may last one, two, or more months, is usually confined to the lateral pharyngeal bands, is accompanied by very slight inflammation, never produces more than slight soreness, in no way affects the child's health, and is, in fact, usually discovered by some casual examination of the throat. Dr. MacDonald has described similar cases, the average duration of which was ten weeks. Some ob-

servers state that fibrinous rhinitis never follows nor is associated with a similar condition in the pharynx, but this statement, although generally correct, cannot be accepted as absolute.

9. *The general symptoms* were in every case unimportant, and this is one of the most marked features of the disease. In the majority of cases close questioning elicited the fact that the children had been somewhat indisposed at the commencement,—slight feverishness at night, headache, languor, diminished appetite, restlessness, &c., having been noticed. These symptoms passed off in a day or two, and were in no case sufficiently severe to confine the child to bed, or to make the mother seek advice. In some cases no history of initial disturbance at all could be obtained, and the children in most cases were allowed to play about or attend school as usual. Until they came under treatment, some of them did not miss a single day's attendance at school.

The insignificance of the general symptoms may also be gathered from the fact that about two thirds of the cases did not come under observation until the disease had lasted three weeks or longer, the chief symptom for which advice was then sought being the local discomfort.

Again, some cases were brought up for quite other reasons,—thus one child attended for a tubercular sinus in the neck, one for a suppurating ear, two were sent to me as probable cases of foreign body in the nose, and two children, sisters of others similarly affected, were brought up at my request to see the other members of the family. In these cases the mothers had noticed the children had a slight cold, but regarded it as of no importance.

The case of the boy aged fifteen commenced whilst he was in hospital, and I am indebted to Dr. Burney Yeo for permission to make use of it. He had bilateral fibrinous rhinitis with some exudation in the post-nasal space and on one tonsil, as above described. The temperature reached 99·8° on the first evening of the affection, but it had occasionally gone up higher than this on other occasions. After-

wards it remained normal throughout the affection, which lasted five weeks. There was no general disturbance of any kind and no sequelæ. This is the more remarkable because of the extent of the local affection, and because the patient was suffering from severe diabetes. The urine contained 2500—3000 grains of sugar per diem, albumen, and occasionally acetone. The boy weighed only 5 st. 4 lbs., and was steadily losing ground.

Even thoroughly competent observers may overlook these cases. A case I have quite recently seen was for eight weeks an in-patient in the surgical ward of a children's hospital, and was considered to have only a bad cold. I was not asked to see him until fourteen weeks from the commencement of the trouble. In another children's hospital also I have recently been informed of a case which those of the staff who saw it pronounced not to be diphtheria, whatever else it might be.

10. *Progress.*—The disease runs a subacute, occasionally almost a chronic course, the duration varying from three to twelve weeks, the average being about five weeks.

11. *Prognosis and sequelæ.*—Some of the children during the course of the disease became rather pulled down and looked pale and languid, but the large majority remained throughout in their usual health. They were kept under observation two to three months, and in none were any paralytic phenomena observed, such as loss of knee-jerk, paresis of palate, &c., although these were regularly sought for.

In every case complete recovery took place, but in two instances there were observed some adhesions between the inferior turbinate and the septum.

12. With regard to the *causation* of the disease, apart from infection, which will be discussed later, I have been unable to make out anything definite. Two of my cases followed severe blows on the nose—a fact to which I should attach slight importance, although Pluder and others have recorded similar cases, regarding the blow as a predisposing cause. Dr. MacDonald considers previous attacks

as a predisposing factor, and I have seen one typical case recurring two successive springs in a young girl. In one of the present cases a relapse occurred after a five months' interval, the first attack lasting one month, the second three months.

Diseases of the nose, such as atrophic rhinitis (of which several instances are recorded), and such affections as measles, scarlet fever, and influenza, must undoubtedly be considered predisposing causes.

True diphtheria may also undoubtedly attack the nose. The affection commences acutely, the nose is completely blocked, and membrane may be seen in it on examination. The discharge is profuse, thick, and purulent, and usually brown from admixture with blood. It is very acrid, producing much irritation of the lip, and is usually peculiarly foetid. Epistaxis is also a common symptom, and occurs quite early in the disease quite apart from traumatism. The general symptoms are all pronounced, the child is quite prostrated and evidently extremely ill. Albuminuria is commonly seen and is often marked. The local affection is always bilateral, and it may spread to the throat as typical faucial diphtheria. The severe general symptoms continue and increase in gravity. Statistics of the North-Western Fever Hospital show that diphtheria affecting the nose is the most dangerous of all forms, the fatal cases being 67 per cent., and this is in general accord with the experience of others.

In the cases which survive, paralytic phenomena are common and severe.

This affection from its very acuteness is rarely seen in the throat departments. Two cases have, however, come under my care during the last year in which a purulent nasal discharge has persisted after an attack of diphtheria. One of these cases had suffered from faucial diphtheria and been treated in a fever hospital for some six weeks. It was brought to me about a fortnight after its return from the hospital with a purulent discharge still continuing from the nose, although no membrane could be seen, and

with paralytic symptoms, viz. weakness of the legs, loss of knee-jerks, and complete palatal paralysis. On its return from hospital one of its brothers had taken diph- theria and had died. This patient was promptly sent back into isolation.

Another case was similar, having been treated at home for a severe sore throat which confined her to bed for about a week. A month later when I saw her there was a purulent discharge from the nose, weakness of the legs, and paralysis of the soft palate. Another child in the same house, a playmate, came to me at the same time with a small membranous deposit in the throat and a discharge from the nose. At the same time the child looked pale and ill, although it was not really bad. Later it developed paralysis. In none of these cases could the diagnosis be in doubt.

Comparing fibrinous rhinitis with nasal diphtheria, we find that the latter is an acute affection characterised by very serious constitutional disturbance, that the prognosis is most grave, and that in cases which survive paralytic sequelæ are common. Fibrinous rhinitis, on the other hand, is marked by the very slight amount or complete absence of general symptoms, the entire absence of all sequelæ, the absolutely good prognosis, and the relative chronicity of the affection. The local signs in nasal diphtheria are more acute and more severe, bleeding is more frequent and occurs early quite apart from trau- matism, the discharge is usually foetid, the affection is bilateral, and may spread to the fauces as typical diph- theria. Fibrinous rhinitis is chronic, bleeding rarely occurs till late and is rarely much, being due to slight traumatism ; the discharge is rarely foetid. The affec- tion is freqently unilateral, and is usually limited to the nasal mucosa. When the throat is involved the appear- ances are characteristic, and distinct from what is usually recognised as diphtheria.

Without denying that intermediate cases may occur, clinical differences between the two affections are com-

monly so well marked that the older observers without exception did not hesitate to pronounce them distinct diseases if the possibility of their identity ever occurred to them.

Bacteriological investigation.—All the cases of this affection coming under my observation during the last fourteen months (thirty-three in number) have been examined bacteriologically, and in all very definite results have been obtained.

In many cases where the nasal discharge was easily obtainable it was examined at once by smearing it on cover-glasses, fixing, and staining with Löffler's solution of methylene blue. In the early cases numerous clumps of typical Klebs-Löffler bacilli were readily distinguished, but when weekly examinations were made the bacilli diminished rapidly in numbers as the discharge became less.

This method is of great use as a means of rapidly forming a diagnosis, and although a negative result cannot be regarded as conclusive, I have never failed to find numerous bacilli in cases where the discharge was profuse.

In every case tubes of Löffler's blood-serum were inoculated with the nasal discharge or membrane, and incubated in the hot incubator (37° C.) for eighteen to twenty-four hours. In all the cases an abundant growth of the typical Klebs-Löffler bacillus was obtained. In fifteen cases the bacillus was found in apparently pure culture, in eighteen cases there were also present staphylococci, streptococci, and occasionally other organisms.

The bacilli were nearly always (twenty-seven out of thirty-three) of the large variety, some exceptionally large, and they showed all the morphological characteristics—bipolar staining, metachromatism, polymorphism, &c.—of the true Klebs-Löffler bacillus.

In some cases cultures were inoculated every week from the nose, and the bacilli were found to persist

throughout the course of the disease, but had often disappeared in a week, and were never found a fortnight after the cessation of the discharge. They retained the same type throughout.

Recently many bacilli closely resembling the Klebs-Löffler bacillus have been described, and I have therefore carried out some experiments to establish the identity of the bacilli found in these cases.

A. The bacilli were obtained in pure culture when necessary by repeated inoculations on serum or by plate-cultures on agar. They were then sown on other media, as agar and gelatine.

In each case the growth of the colonies and the cover-glass preparations from them were similar to those of the true Klebs-Löffler bacillus. On litmus sugar-agar a distinct acid reaction was obtained.

B. *Experiments on animals.*—In twenty-three of the cases a pure culture of the bacillus was inoculated into peptone beef-broth and incubated for forty-eight hours at 37° C. If, after incubation, the culture on examination proved still pure, a portion of it was injected subcutaneously into a guinea-pig. The doses used varied. In seven cases a dose of 2 c.c. was given, which proved fatal in six instances within two days, and in the remaining instance within three days. In nine cases doses of 1·5 c.c. were given, and six guinea-pigs died within two days, two within three days, and one within four. In two other instances doses of 1 c.c. proved fatal in thirty-six hours each. In the last five cases smaller doses of 0·5 c.c. were used. One guinea-pig died in about twenty-four hours, two in forty-eight, and two in three days.

In every case post-mortem examination showed intense hæmorrhagic œdema at the site of injection, and apparently the longer the animal survived the more marked was this local reaction.

In one case a rabbit was tracheotomised and the tracheal mucous membrane rubbed with a pure culture

of the bacillus. The animal died on the third day. The whole neck and part of thorax was extremely œdematous with small scattered hæmorrhages, and there was a membranous exudation lining the whole trachea and extending down to the bronchi.

Thus the virulence of the bacilli, as tested on animals, was found to vary, but in every case was considerable, and this is a further proof of the identity of the bacillus with the true Klebs-Löffler bacillus.

In ten cases the virulence of the organisms was not tested. In eight this was due to the fact that the bacilli were not easily obtainable in pure culture. Although they appeared in many instances to be at first almost pure, other organisms grew abundantly in the sub-cultures, and the Klebs-Löffler bacilli rapidly disappeared. In some cases even by plate-cultures no bacilli could be obtained. This fact was also observed by Ravenel in some of his cases, and led him to believe that the bacilli were not very virulent, and especially were less likely to spread infection. As a rule, however, the bacilli showed their vitality when grown on blood-serum by rapidly crowding out other organisms, pure cultures being readily obtainable simply by the process of making repeated sub-cultures. I kept some of my cultures alive over seven months, transferring them to fresh tubes not oftener than once a month or six weeks.

Nearly all the failures occurred during last summer, when I was unable to give daily attention to the work, and I attribute the fact not so much to the feeble vitality of the Klebs-Löffler bacillus as to the increased growth of the associated organisms, sarcinæ, &c., while the cultures were laid aside. In no case did a pure culture die out if re-inoculated about every four weeks.

c. *Toxin-producing experiments.*—Dr. Macfadyen suggested that I should test the toxin-producing powers of the bacilli. For this purpose pure cultures obtained from typical cases, No. 1 of bilateral and No. 2 of unilateral fibrinous rhinitis, were taken, together with a third culture,

No. 3, obtained from a case of severe faucial diphtheria to act as a control.

Flasks containing about 70 c.c. of peptone beef-broth were inoculated from these cultures and grown for sixteen days. These cultures were then examined, and no foreign organisms being discovered they were sterilised by filtration. To the clear filtrate ½ per cent. carbolic acid was added. The toxins being thus obtained, the following experiments were made. A dose of 0·5 c.c. of toxin No. 1 (prepared from the bilateral fibrinous rhinitis case) killed a large guinea-pig in thirty-six hours, a dose of 0·1 c.c. of the same toxin being fatal in the same time. The latter result was so unexpected and indicated such a powerful toxin that the experiment was repeated on three occasions, always with a like result.

Doses of 0·5 c.c. and 0·1 c.c. of toxin No. 2 prepared from the case of unilateral fibrinous rhinitis were fatal in thirty-six hours and four and a half days respectively. Similar doses of the No. 3 toxin (the diphtheritic one) killed in three and a half and four and a half days respectively. Doses of 1 c.c. of No. 2 and No. 3 were also given, and both proved fatal in thirty-six hours. Thus one of the toxins obtained was at least equally virulent, and one much more so, than that obtained from the diphtheria bacilli which was used as a control.

D. *Experiments with antitoxin.*—Experiments were next performed to determine if these toxins were neutralised by the diphtheria antitoxin as sold for remedial purposes. Of toxin No. 1 doses of 0·5 c.c. were injected into two guinea-pigs, together with 1 c.c. and 0·5 c.c. of antitoxin. The animals lived for six and eighteen days respectively. Further, a dose of 2·5 c.c. of this toxin, that is twenty-five times the dose shown to be fatal in thirty-six hours, was injected into a guinea-pig with 0·25 c.c. of antitoxin, and the animal lived for thirteen days. Of toxin No. 2 doses of 1 c.c. and 0·5 c.c. were injected with similar doses of antitoxin : the animals lived nine days. With a dose of 0·1 c.c. of toxin and 0·1 c.c. of antitoxin

the animal survived.   Toxin No. 3 (the control) was used in doses similar to No. 2, and the animals survived eleven days.

Although these experiments are not entirely satisfactory they conclusively prove the neutralising effects of the diphtheria antitoxin on the toxins obtained from the bacilli of fibrinous rhinitis.   The death of the animals in some of the cases is probably due to the fact that such large doses of toxin were given, the antitoxin acting as a certain antidote only when the minimum fatal doses of toxin are used.

Similar results were obtained when antitoxin was injected together with living cultures of bacilli.   Thus in one case 0·5 c.c. of forty-eight hours' broth culture was fatal in twenty-four hours.   Given with 0·5 c.c. antitoxin the animal survived six days.   In another case the same dose of broth culture killed in three days; given with antitoxin the animal survived.   In a third case 0·5 c.c. of broth culture killed in three days; given with antitoxin the animal survived.

The results of these experiments, briefly stated, show that in cases of fibrinous rhinitis an organism is constantly found in extraordinarily large numbers which resembles, morphologically and by its growth on various culture media, the true Klebs-Löffler bacillus.   The bacillus is of varying but usually great virulence, is capable of producing virulent toxins, and these toxins as well as cultures of the living bacilli are neutralised by diphtheria antitoxin.   The bacilli inoculated on the mucous membrane of a rabbit's trachea produced an extensive membranous exudation, resembling that of human diphtheria.

These experiments place beyond doubt the identity of the bacilli with the true Klebs-Löffler bacillus, and also show that the mildness of the affection in no way depends upon the slight virulence or feeble toxin-producing powers of the organisms.

Thus, as to the identity or non-identity of fibrinous

rhinitis with diphtheria, the bacteriological evidence is in direct opposition to the clinical. Further evidence of the greatest importance can be obtained by a careful clinical and bacteriological examination into the surroundings of these cases, and of the persons with whom they come in contact. The great facilities this affection offers for a wide-spread diffusion of infection is apparent when we consider that the nasal discharge in most of the cases was profuse, and was constantly dripping from the nose or being sneezed about over everything; that it was swarming with bacilli, that it lasted as a rule several weeks, and that owing to the absence of general symptoms the children were not seen medically until two or three weeks from the commencement of the disease. For the same reason the affected children attended school, and played with other children as usual. Belonging to the poorer classes, a whole family with many children often lived in one room, necessarily in close contact, and to further aid the spread of infection the mother would use her own handkerchief to the noses of all the children.

This inquiry into the patient's surroundings falls naturally into three headings.

1. Could these cases of fibrinous rhinitis be directly traced to diphtheritic infection ?

2. Was there found a wide-spread diffusion of the bacilli as is above presumed ? and—

3. Did outbreaks of true diphtheria arise in connection with these cases ?

In regard to the first point I could only find one case in which diphtheria might have been the cause of infection, and this is somewhat doubtful. The patient's father was said to be suffering from diphtheria, but the patient, a child aged 1 year and 10 months, was, at the time I saw it, and had been for three weeks previously, living with another family, and had not been in any communication with its father. I cannot determine if the father really had true diphtheria or such

a sore throat as I shall later describe, but the child showed no sign of it, nor did it occur amongst the other children (five) with whom this patient was living. Of course diphtheria was all the time very prevalent in London, but my cases did not correspond in frequency with the reported cases of diphtheria (the numbers of course are very small) ; nor was there any special incidence of diphtheria in the schools these children attended.

In three cases, and in a fourth which I have lately seen, there was a definite connection with scarlet fever. These cases and the case of diphtheria above mentioned may quite well have been mere coincidences.

These results are confirmed by nearly all other writers, including the whole of the older observers ; and amongst the more recent ones, Ravenel in ten cases, Gerber and Podack in six cases, Park and Abbott six and three cases, although all these latter believe firmly in the diphtheritic nature of fibrinous rhinitis.   Pluder in six cases found one in which there had been diphtheria six weeks previously in the house then occupied by another family. Johannessen reports one case in connection with an outbreak of diphtheria in a children's hospital, and there are a few others reported without any details by Eeman, Hunt, &c.   Most observers state that they have especially endeavoured to obtain evidence of such infection, and have been able to exclude it.   It may, I think, be concluded that diphtheria very rarely gives rise by direct infection to fibrinous rhinitis.   With regard to the second and third points I obtained such striking results that some of the cases must be dealt with individually.

Cases 1, 2, and 3 occurred in the persons of three children in one family, two girls and a boy. The boy, aged 6, was first attacked, and a week later his two sisters, aged 4 and 9 years, were attacked. All three patients suffered in exactly the same way with typical bilateral fibrinous rhinitis which lasted six to eight

weeks. About five weeks from the commencement of the affection the mother had a sore throat, which lasted two to three days, and was not at all severe. She was not laid up with it, and had no subsequent ill effects. The other people in the house, a father and a two-year-old daughter, remained well throughout.

There was no history of diphtheria in connection with these cases either before or after. It was not prevalent in the district, and there had been no case at the school the children attended.

CASES 10 and 11.—A girl aged 4½ was brought to me with a small sinus in the neck. She was noticed to have a discharge from the nose, and on inquiring the mother informed me she had noticed the child had a cold in the head for the last three weeks. It was seen to be a typical case of bilateral fibrinous rhinitis with complete absence of general symptoms. Virulent Klebs-Löffler bacilli were found both in the nose and in the pus from the sinus. The child's sister was said also to have a slight cold, and being brought to me was found to be similarly affected. Three adults in the house remained well.

CASES 28 and 29.—Two sisters, aged 6 years and 13 months, with bilateral fibrinous rhinitis. The elder child was affected a month before the younger, and had attended school throughout, only coming under observation about the fifth week of the disease. The father, mother, and another sister, aged 2½, living with them, remained well. Three other families with nine children living in the house, and all remained well.

CASES 32 and 33.—A girl aged 5½ was sent to me as a case of foreign body in the nose, unilateral nasal discharge having been present about fourteen days. Under chloroform a large thick piece of membrane was removed from the affected nostril. A week later the child's sister, aged

ten months, was brought to me similarly affected with unilateral fibrinous rhinitis. The other people in the house, all adults, remained well.

Thus out of my thirty-six cases nine occurred in four families. In three other instances other children in contact with these cases were said to have sore noses or colds in the head, but I did not see any of them.

In connection with the above group of cases it was noted that in one instance the mother of the child had a sore throat. Other instances are as follows :

CASE 9, a boy aged 15, in King's College Hospital, under the care of Dr. Burney Yeo, who was treating him for severe diabetes as above described. Through the courtesy of the staff I was able fully to investigate the surroundings of this case. All the patients who had been some days in the ward and the nurses were examined, and cultures made from the tonsils. They numbered in all thirty-two. Of these twenty-one showed no bacilli on cultivation, and suffered from no throat affection. Of the remaining eleven, two showed numerous colonies of the pseudo-diphtheria bacillus of Von Hofman in pure culture, and without any symptoms of sore throat. The remaining nine gave cultures of typical Klebs-Löffler bacilli. These cases are briefly as follows :

i. Boy aged 12, under treatment for pericarditis, but now allowed to get up. He had no sign or symptom of sore throat. Cultures showed numerous colonies of pure Klebs-Löffler bacilli.

ii. Man aged 45, allowed to be up in ward. Had no sign of throat trouble. Numerous colonies of Klebs-Löffler bacilli.

iii. Adult. Had had no sore throat. One colony of Klebs-Löffler bacilli obtained.

iv. Nurse, who said her throat was not and had not been sore, but the tonsils appeared slightly red and enlarged. She remained constantly on duty. Numerous

colonies of Klebs-Löffler bacilli obtained in pure culture. Six weeks later this nurse had a sore throat, but no bacilli could then be found.

v. Boy, under treatment for pericarditis, says throat has not been sore, and there is nothing to see on examination, but the glands at the angle of the jaw have been slightly enlarged and tender the last two days. Numerous colonies of pure Klebs-Löffler bacilli obtained.

vi. Boy had a slight sore throat lasting two days only, with swelling and redness of the tonsils. Fairly numerous colonies of Klebs-Löffler bacilli, with other organisms, obtained on cultivation taken two days after the sore throat had got well.

vii. Boy aged 18, suffering from advanced diabetes. White patches of exudation seen spreading over both tonsils, and glands at angle of jaw enlarged. Temperature reached 101° F. once only on the first day. There was no general disturbance and no sequelæ. He was quite well by the fourth day.

viii and ix. Two nurses with rather severe sore throats and some general disturbance at first. Clinically follicular tonsillitis with somewhat profuse exudation. Numerous colonies of Klebs-Löffler bacilli obtained in each case. The general disturbance subsided in a few days, and there was no subsequent trouble.

Thus we have here among thirty-two people exposed to infection from one case of fibrinous rhinitis, twenty-one free from any sign of sore throat or bacilli, at least on the one occasion on which they were examined ; two in whom pseudo-diphtheria bacilli were found without any throat trouble ; three in whom Klebs-Löffler bacilli were found, in two of which they were numerous, and all three without throat trouble ; and six cases in which more or less sore throat occurred in conjunction with numerous bacilli, generally in pure culture. One case of sore throat of a similar kind had occurred just before in the ward, but had not been examined bacteriologically. Excluding

this one we have six cases of sore throat and one case of fibrinous rhinitis *plus* sore throat, all arising in association with each other, some of the patients being already in a very depressed general condition, and yet none of them presented any feature clinically characteristic of diphtheria.

The results obtained in this case I have quoted fully, as I was able to make such a complete inquiry ; but they are confirmed by those obtained in other cases, some of which are briefly as follows :

CASE 7.—Boy aged 7½, suffering from bilateral fibrinous rhinitis and the typical form of pharyngeal affection which has been above described as sometimes associated with this disease. The affection lasted ten weeks. The mother and three children lived with this boy, and all remained well but one sister. The latter, aged 12, had a sore throat lasting two days, which was not severe enough to prevent her attending school. She was brought to the hospital four days after the attack at my request. Nothing abnormal could then be detected in the throat, but typical Klebs-Löffler bacilli were obtained from the tonsils by cultivation on serum. They formed numerous colonies in almost pure culture, and proved to be of full virulence, 1·5 c.c. of forty-eight hours' broth culture killing a guinea-pig in forty-eight hours.

CASE 8.—Boy aged 5, with bilateral fibrinous rhinitis, lived with his father, mother, and two other children. The father and one child remained well throughout, and no bacilli were obtained from their throats. The mother had a sore throat about the time the boy was first attacked. I did not see her, but as far as I could ascertain it was only slight, and caused her no subsequent trouble. The other child, aged 2½, had a similar sore throat at the same time, which lasted two to three days. When I saw the family three weeks later this child appeared quite well and the throat was normal, but

cultivations from the tonsils yielded a numerous growth of true Klebs-Löffler bacilli. Four other people in another flat of the same large dwelling-house were said to have had sore throats which were not diphtheritic, and did not seriously indispose them. I saw none of them.

CASE 12.—Boy aged 5, with unilateral fibrinous rhinitis lasting one month, and recurring again in five months and lasting three months. He lived with his father, mother, and two brothers, and one of the last had a slight sore throat two months before being seen. Cultivations from the mother's throat showed no bacilli; from the two brothers the pseudo-diphtheria bacillus was obtained.

CASE 13.—Boy aged 3, with unilateral fibrinous rhinitis, came under observation on the fourteenth day of the disease. The mother and one sister had had sore throats, the other sister remained well. No bacilli were obtained from any of them.

CASE 15.—Boy aged 4, a rather severe unilateral case. Of the father, mother, and three other children one only of the last had a slight sore throat. Cultivations made from the throat showed pure cultures of Klebs-Löffler bacilli in the case of the mother and one sister, and yielded negative results in the other cases.

CASE 16.—Girl aged 7, with bilateral fibrinous rhinitis with much discharge, was seen on the fourteenth day of the disease. The child's mother and sister had slight sore throats a week before the child was seen, and the only other person in the house, a man aged 19, had a slight sore throat a week later.

CASE 19.—Girl aged 6, a slight unilateral case, lived with her mother and two sisters. All remained well, but typical large Klebs-Löffler bacilli were obtained from the throat of one child.

CASE 17.—Boy aged 2, whose disease had lasted six weeks; the mother had had a slight sore throat, and the sister had had a little running at the nose. The father and three other families in the same house all remained well.

CASE 20.—Girl aged 4, first seen on eighteenth day. The mother and one sister had had slight sore throats, and the baby was brought to me with slight tonsillitis. The father of the child and two other families containing nine children living in the same house all remained well.

In other instances complete absence of infection was noted. Thus Case 4, a child aged 3, came under observation in the third week, and the affection lasted three weeks longer. The discharge from the nose was profuse. The child lived in one room with the mother and four other children, all of whom were said to be quite well throughout. There were other similar cases, but it is possible that, when the family were not seen by me, mild cases of sore throat were overlooked.

Another case which I have quite recently seen is remarkable.

A boy aged 5½, who had been affected apparently about two months, was admitted to the children's hospital with impetigo on the face and corneal ulcers. These latter were treated and the nose was syringed. He was in hospital eighteen days, then in convalescent home eleven days, and then again in hospital five weeks. The disease lasted all this time, and being unrecognised no precautions as to isolation were adopted. During this time over thirty children came into more or less direct contact with him, and yet there was no case of diphtheria or sore throat in the ward apart from two cases of scarlet fever. These latter led to the closing of the ward. I did not see the boy till the end of his stay in hospital about the fourth month of the disease, when I found typical bilateral fibrinous rhinitis, and obtained by

culture a pure growth of Klebs-Löffler bacilli of full viru-
lence. In this case the disease lasted over four months,
and for nearly a month he attended school, a month he
was at home and with two other families, and two months
he was in hospital. Nowhere was there another case of
sore throat.

Summing up these results, we find that twenty-five
cases of sore throat occurred in direct connection with
eleven families affected with fibrinous rhinitis. Many of
these were seen by me, and presented none of the clinical
symptoms of diphtheria, although from all of them the
diphtheria bacillus was isolated. The other cases were
all slight, and as far as the history obtained goes were
not diphtheria. Also in four instances a case of fibrinous
rhinitis was the apparent starting-point of the same
affection in others, in one family there being three cases
of it. Finally, the diphtheria bacillus was frequently
found in the throats of healthy people in contact with
these cases.

From these results we may conclude that fibrinous
rhinitis is an infectious disease, and that it has a special
tendency to reproduce itself. This fact has been ex-
pressly noted by Ravenel, two of whose ten cases were in
sisters. Seifert, Chapin, and Abbott have recorded similar
instances. Other observers have more or less incidentally
mentioned that sore throats occurred in connection with
these cases, and Pluder has especially dwelt on this point
as proving the diphtheritic nature of the disease. He
records six cases, in three of which spread of infection
occurred.

(i) Boy aged three, with bilateral fibrinous rhinitis
and small patch on one tonsil. Had four sisters exposed
to infection ; two had bilateral exudation on the tonsils,
and one had angina without exudation. It is noted that
in all cases general disturbance was absent. All showed
virulent bacilli.

(ii) Boy aged five. Had typical bilateral fibrinous
rhinitis, and another two days later had hemp-seed sized

patch on one tonsil. Cultures from nose and throat showed virulent bacilli. There was a history of diphtheria in the house six weeks before this family entered it.

(iii) Young housemaid with mild naso-pharyngeal diphtheria. Next day young child in house had bilateral fibrinous rhinitis and faucial congestion. Both showed virulent bacilli, as also did cultures from throats of three healthy people in house.

In other cases the bacilli were found in the throats and noses of exposed persons, but without any morbid lesion.

Pluder considers all the above throat infections as diphtheria, but it seems to me that clinically they were distinct, and would not have been considered diphtheritic but for the bacteriological examination.

The results of other observers in the main agree with the above, and, taken all together, go far to show that fibrinous rhinitis, although it is very infectious and gives rise to throat affections, has slight if any tendency to give rise to diphtheria. Ravenel was unable to record such a result in his ten cases, although he states that he particularly sought for it; in fact, the only recorded cases I have found are those of Concetti, Gerber and Podack, Hunt, and Eeman.

Concetti describes his case as primary nasal diphtheria, and this is a more correct description of it than fibrinous rhinitis would be, for it differed from the latter in essential particulars, and was followed by paralysis. Similar remarks apply to the case recorded by Gerber and Podack. Hunt and Eeman simply relate cases without detail.

I have gone very fully into this point, as it seemed to me of the greatest importance. The affection seemed to offer such exceptional facilities for the spread of infection—and this spread of infection does indeed take place—that it seems very remarkable, if the disease is diphtheritic, that cases of unmistakable diphtheria should not occur in connection with it. And yet in thirty-six carefully observed cases I have been unable to find a

single instance of such occurrence, and the few recorded instances seem doubtful. Without, therefore, denying its possibility, its occurrence must be admitted to be extremely rare and at present not authenticated.

To obtain some information as to the diffusion of the bacillus of diphtheria, and to control my results, I made cultivation experiments from the noses of a hundred different individuals. This experiment lasted from September 29th to October 10th, and included every case of a child attending my out-patient practice and twenty-five cases from Dr. Sutherland's medical clinique. All the patients were under twelve years of age, and many were infants. There was no history in any of them of exposure to diphtheritic infection. The children were attending for a large variety of complaints, but of those in my own practice (seventy-five) more than half were cases of adenoids, and in other respects enjoyed good health. None of the patients were seriously ill. A few of the cases (four) had atrophic rhinitis, and many of them had a slight running at the nose.

The cultures were made in the usual way. Small sterilised swabs of wool on wire were inserted into the nostril and then rubbed over the surface of Löffler's blood-serum. This was incubated for eighteen to twenty-four hours at 37° C., and then examined by cover-glass preparations. In nearly every case a numerous growth was obtained. The results as regards the diphtheria bacillus were as follows :—In five cases a pure culture of the Klebs-Löffler bacillus was obtained, and in eight other cases this organism was found together with cocci, sarcinæ, &c. In sixteen cases a bacillus resembling more or less closely the diphtheria bacillus was obtained in pure culture, and in thirty-six other cases in more or less mixed growth. Further experiments have proved this bacillus to be the pseudo-diphtheria bacillus of Hofman.

Thus we find that in cultivations from the nose in children the Klebs-Löffler bacillus is found in about 13 per cent., and Hofman's bacillus in 52 per cent.

Johannessen examined the throats of twenty healthy children in the ward of a hospital in which a case of diphtheria had occurred. He found virulent diphtheria bacilli present in three cases. Later, another case of diphtheria having occurred, he found the bacilli present in four out of eighteen healthy throats.

Aaser in similar circumstances, an outbreak of diphtheria in a soldiers' barracks, found the Klebs-Löffler bacilli in seventeen out of eighty-nine healthy throats, that is 19 per cent. In an outbreak amongst some children, bacilli were found in 20 per cent. Maude reports finding bacilli in eighty-nine out of 214 healthy people exposed to infection, that is in 41 per cent. Maude also reports that diphtheria bacilli were present in forty out of 148 cases of angina said not to be diphtheritic, that is in 27 per cent. Müller finds the Klebs-Löffler bacilli present in twenty-seven cases out of 100 healthy children with normal throats—curiously. the exact proportion in which Maude found them present in cases of non-diphtheritic angina. Thomas found the diphtheria bacillus (usually non-virulent) in nasal discharge in eighty out of 326 cases examined, that is 24 per cent.; and Vassant found them in similar circumstances in twenty-six cases out of 100 examinations.

These investigations show conclusively that the Klebs-Löffler bacillus is found commonly in the normal throats of healthy people who have not been exposed to diphtheritic infection, although not quite so commonly as in those who have been exposed to it; that the bacillus is found just as frequently in cases with normal throats as in cases with non-diphtheritic angina; that the bacillus commonly occurs in the healthy nose, and is found in about 25 per cent. of all cases with any form of nasal discharge.

Conclusions.—It has been claimed that just as the tubercle bacillus when found in any morbid condition is the decisive test of the tubercular nature of the affection,

so every morbid condition in which the Klebs-Löffler bacillus is found must be pronounced to be diphtheria whatever the clinical appearances. Without agreeing with this dictum it must, I think, be allowed that if the Klebs-Löffler bacillus is *constantly* found in an affection clinically characterised by the formation of membrane this affection is a form of diphtheria. Fibrinous rhinitis, however, presents in its clinical aspects and associations such marked differences from diphtheria that I think the distinction is worth maintaining. As Osler puts it, the Klebs-Löffler bacillus gives rise to two distinct affections in the nose, and I think it might be added in the pharynx also. And we adopt a similar course with regard to tubercular affections. Thus lupus of the larynx is clinically entirely distinct from laryngeal phthisis, and yet both are indisputably tubercular.

Similarly, if we admit with the large majority of recent observers that the *Streptococcus erysipelatis* of Fehleisen is identical with the *Streptococcus pyogenes*, we must still admit that erysipelas, cellulitis, and an acute abscess are distinct clinical affections, although all due to the invasion of the tissues by the same organism.

With regard to the latter organism, we know that Fehleisen, inoculating pure cultures of it obtained from a case of erysipelas into human beings, produced always erysipelas, and not other affections ; and also conversely that inoculations into the skin of streptococci from an abscess, either experimentally or when an acute abscess containing them is opened, never produce erysipelas.

For these reasons Watson Cheyne among others has maintained that the organisms are distinct, and my own observations on fibrinous rhinitis, and more especially on the diseases to which it gives rise, which are really a natural series of experiments on human beings, might be used as an argument against the specificity of the Klebs-Löffler bacillus. Hanseman and others have indeed argued thus simply from the clinical differences between fibrinous rhinitis and diphtheria, and from the wide-spread

distribution of the bacilli in healthy throats.   The arguments in favour of the Klebs-Löffler bacillus are, however, so overwhelming that it seems unnecessary to pursue this line of argument, and more profitable to attempt some explanation of the fact that it does give rise to two distinct affections.

1. The most obvious explanation would be that the individuals attacked are more or less immune to diphtheria.   It might be urged that a local lesion is necessary to enable the bacilli to obtain a footing, that the nose is an unfavorable site for them, or that absorption of bacillary products takes place very slowly from the nasal mucous membrane, or that there is constitutional immunity in these cases.   It has been urged that the absorption of toxins takes place slowly in this affection, and allows neutralising antitoxins to form in the blood sufficiently rapidly to prevent much constitutional disturbance. The absence of a local lesion may account for the fact that the bacilli are frequently found in the throat or nose without apparently producing any symptoms, but it cannot be the explanation of the absence of general symptoms, &c., when the bacilli have obtained sufficient foothold to produce a membranous exudation.   Again, there seems no sufficient reason for supposing that absorption from the nose is slow or slight, but even granting this, in some of my cases the disease attacked the pharynx, and it spread to the pharynx in others, and we know that absorption takes place readily enough in this region.   It is true that the affection of the throat was of a peculiarly superficial, pseudo-membranous character, but a satisfactory explanation must account for this fact.   Most important of all, the affection, although giving rise to wide-spread infection did not give rise to true diphtheria, and we can hardly suppose that all the persons exposed to infection were more or less immune.   They included, as we have seen, many feeble individuals in the ward of a general hospital, the surgical ward of a children's hospital, a convalescent home for children, and many large and young families.

The above theories failing, we must seek an explanation in some modification of the infecting organism or organisms. Many observers have said that the bacilli found are of feeble virulence, that they do not *kill* guinea-pigs (Abbott), or that cultures of them die out even when sub-cultures are frequently made (Park and Ravenel). Later observations and my own prove that, although the virulence of the organisms varies somewhat, in the great majority of cases it is well marked or even extreme. Further, I have shown that cultures of the bacilli can be kept alive many months even when sub-cultures were made at intervals of five to six weeks.

I have also shown that the absence of general symptoms is not due to the deficient toxin-producing powers of the bacilli, and therefore must be due to the non-absorption of the toxins formed. This latter may be, and probably is, due to the fact that the local changes, the necrosis of the tissues, is less deep in fibrinous rhinitis than in diphtheria.

Other organisms, and especially the pyogenic streptococci and staphylococci, have been considered active associates with the Klebs-Löffler bacillus in the production of severe diphtheria, and their absence to account for the mildness of fibrinous rhinitis. But these organisms are frequently found in fibrinous rhinitis, and also in the milder as well as in the severer forms of diphtheria. These pyogenic organisms, however, vary much in virulence, and can hardly be supposed to play no part in the disease. It seems possible that in diphtheria some organism is present which is not found in fibrinous rhinitis; that the latter is a simple Klebs-Löffler bacillus, and the former a mixed infection. Or, what comes to much the same thing, the organisms associated with the Klebs-Löffler bacillus, possibly the pyogenic cocci, are much more virulent in the one case than in the other. If this explanation be not the true one, the alternative seems to be that the Klebs-Löffler bacillus found in fibrinous rhinitis is not of full virulence,—that is, that the animal

experiments detailed above do not give reliable results when applied to man. Although this may be possible, one would hardly expect it, and the foregoing explanation seems to me the more likely one. But the question must remain undecided at present.

I have not touched on the treatment of fibrinous rhinitis, but the question of isolation must be considered. Nearly all observers have insisted on the absolute necessity of rigid isolation, but it is difficult to see how this can be effectually carried out. As I have shown, the patients are well enough to attend school throughout the disease, and as a rule come under observation very late in its course. Probably many other cases are not seen by a medical man at all, or if seen not recognised. Of course after they have been seen they should, in the present state of our knowledge, be isolated. Fortunately, however, the neglect of this precaution does not seem to be very harmful.

Constantly in the course of this investigation the question of the diagnostic importance of the presence of the Klebs-Löffler bacillus has arisen. The bacillus is found not only in healthy noses, but commonly in noses in association with a slight discharge. These cases surely cannot be regarded as diphtheria. If, however, they must be so considered, and means of isolation adopted, it is quite obvious nothing but a systematic and frequent bacteriological examination of the nose of every child would suffice for the detection of the cases. Neither do I think that every case of sore throat and bacilli is necessarily diphtheria. In many cases I have seen, the soreness of the throat has lasted but a day or two, with no general symptoms; in other cases there has been a slight redness; in some all soreness has been denied. In other cases there has been slight exudation, white points varying in size from a pin's head to a hemp-seed, without any inflammatory signs. Such cases would have passed unnoticed except by systematic bacteriological examination, and if they are diphtheria it seems to me that every case

in which the bacilli are found in the throat must be so considered. For it is probable that the bacilli if found in any quantity in a throat are living and multiplying there, and what degree of change must we consider morbid ? Even if the throat seems perfectly normal we cannot be quite positive, or assert that it was normal the day before.

Further, having regard to the wide-spread diffusion of the Klebs-Löffler bacillus in healthy throats, it is only to be expected that we should sometimes find it in cases of sore throat having nothing to do with diphtheria, and in two whole series of cases by different observers, in different continents, the percentage of cases in which the bacillus was found was curiously enough exactly identical, viz. 27 per cent., one observer dealing entirely with healthy throats, the other with clinically non-diphtheritic angina

It seems to me that, in the present state of our knowledge, we are not justified in concluding that cases of mild sore throat without general symptoms, or any definite clinical sign of diphtheria, are diphtheria, even if the specific bacillus be present, unless they occur in direct connection with other cases of true diphtheria.

The bacteriological work was carried out at the British Institute of Preventive Medicine, where all the various culture media, animals, &c., were provided, and every facility for working was given me. For this and for much advice I have to thank the director, Dr. Macfadyen. I have also to acknowledge my great indebtedness to Dr. R. T. Hewlett, assistant bacteriologist to the Institute, who has freely given me much time and the benefit of his great experience. He has constantly assisted and advised me in every stage of my work, and has performed many of the experiments for me.

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No.	Name.	Age and sex.	Date.	Previous duration of symptoms.	Character of discharge.	Part affected.	Throat.
1	S. S.	M., 6	Aug., 1896	14 days	Purulent, rather profuse	Bilateral; whole nasal mucosa	Not affected
2	L. S.	F., 4	„	7 days	Do.	Do.	Do.
3	M. S.	F., 9	„	14 days	Do.	Do.	Do.
4	E. H.	F., 3	Sept., 1896	21 days	Thick, " mattery "	Large piece of membrane in *one* side	Do.
5	F. B.	M., 3	„	14 days	Profuse, clear	Large piece of membrane *one* side only	Do.
6	A. F.	F., 5·	Oct., 1896	21 days	Thick, " mattery "	Bilateral; whole mucosa	? Slightly at beginni
7	T. R.	M., 7½	„	8 days	Much, clear	Do.	Had tons litis lasti 14 days
8	A. B.	M., 5	Nov., 1896	21 days	Do.	Do.	Not affec
9	G. E.	M., 15	Dec., 1896	2 days	Do.	Do.	Exudation tonsils describe
10	R. C.	F., 4½	Jan., 1897	14 days	Very little, mucous	Bilateral, but only shreddy patches	Not affec
11	J. C.	F., 2½	Feb., 1897	10 days	Do.	Do.	Do.
12	G. T.	M., 5	„	?12 wks.	Do.	Unilateral patch of membrane	Do.
13	W. N.	M., 3	Mar., 1897	14 days	Do.	Unilateral	Do.
14	A. R.	M., 8	„	21 days	Purulent	Bilateral, whole nasal mucosa	Do.
15	V. L.	M., 4	June, 1897	4 days	Do.	Unilateral	Do.
16	G. R.	F., 7	July, 1897	14 days	Do.	Bilateral, whole nasal mucosa	Not affec

[1] Dose of 48-hour culture give

General symptoms.	Duration.	Organisms found.	Virulence of the K.-L. bacilli.[1]	Family surroundings of patient.
Slight head-ache, languid, &c.	6 weeks	No cultures	—	One family. The mother had slight sore throat; father and other sister remained well. Scar let fever at the school.
Do.	4 weeks	Do.	—	
Do.	8 weeks	Do.	—	
Languid and irritable	6 weeks	Pure large K.-L.	1·5 c.c. in 36 hours	Mother and 4 children remain well.
Very slight	5 weeks	Do.	2·0 c.c. in 36 hours	Father and mother remained wel
Headache for one day, but attended school all through	6 weeks	Do.	1·5 c.c. in 36 hours	Mother and 1 brother remain well (source of toxin No. 1).
Not really ill, but general in-disposition; pale, &c., all through	10 weeks	Large K.-L. and strepto-cocci	1·5 c.c. in 2 days	One sister aged 12 had sore thro for one day and virulent K.- found a week later; mother a 1 sister remained well.
Slight at beginning only	6 weeks	Large K.-L. and few cocci	1·0 c.c. in 2 days	Six people in house had slight so throats.
Very slight	5 weeks	Do.	Do.	Thirty-two people exposed. normal, 6 had sore throats a K.-L., 3 K.-L. only, and pseudo-diphtheria bacilli only.
None	3 weeks	Large K.-L. pure	1·5 c.c. in 2 days	Patients are sisters. 3 adults house were well (K.-L. also fou in sinus in neck of R.C.)
Do.	3 weeks	Short K.-L. pure	1·5 c.c. in 4 days	
Do.	14 weeks	Do.	1·5 c.c. in 3 days	Two adults well; 2 brothers h pseudo-bacilli, 1 had a so throat. Similar attack 5 mont ago.
Do.	3 weeks	Large K.-L. mixed	1·5 c.c. in 2 days	Mother and 1 sister sore thro 1 sister well.
Pallor and slight indisposition at first	10 weeks	Do.	1·5 c.c. in 3 days	Two adults remained well.
Slight "feverishness" at first	4 weeks	Do.	Not tested; bacilli died out	Father, sister, and brother we mother and sister had K.- only; 1 brother had sore thro
Slight at first	6 weeks	Large K.-L. mixed	1·5 c.c. in 2 days	Two adults and 1 sister had so throats.

he time required to kill a guinea-pig.

No.	Name.	Age and sex.	Date.	Previous duration of symptoms.	Character of discharge.	Part affected.	Throat.
17	N. V.	M., 2	July, 1897	6 weeks	Thick, slight	Bilateral in patches	Slight at fi
18	L. T.	F., 2½	,,	14 days	Much, clear	Do.	Not affecte
19	L. A.	F., 6	Aug., 1897	9 days	Thick, slight	Right side only, patch	Slight at fi
20	L. U.	F., 4	,,	18 days	Purulent	Bilateral	Slight patc tonsillitis
21	G. W.	M., 11	,,	? 6 weeks	Crusty and thick	Bilateral patches	Not affecte
22	A. P.	F., 8	,,	14 days	Much, clear	Bilateral and all nose	Do.
23	H. E.	M., 6	Sept., 1897	?	Do.	Do.	Do.
24	E. C.	F., 5	,,	7 days	Do.	Bilateral shreddy patches	Redness
25	F. A.	F., 6	,,	10 days	Do.	Do.	Not affect
26	M. P.	F., 7	,,	14 days	Do.	Bilateral and all nose	Do.
27	L. B.	M., 8	,,	21 days	Do.	Do.	Do.
28	E. G.	F., 1 1/12	Aug., 1897	5 weeks	Thick, slight	Do.	Do.
29	L. G.	F., 6	,,	1 week	Do.	Do.	Do.
30	D. S.	F., 3½	Oct., 1897	4 weeks	Thick and bloodstained	Do.	Do.
31	H. M.	M., 5	,,	3 weeks	Much, thick	Do.	Do.
32	L. E.	F., 5½	,,	2 weeks	Thick, slight	Unilateral	Do.
33	D. E.	F., 1 10/12	,,	7 days	Clear, slight	Do.	Do.
34	F. L.	M., 6	,,	14 days	Clear at first, later thick	Bilateral	Slight fi 2 days
35	H. S.	M., 4	,,	14 days	Do.	Do.	None
36	L. W.	F., 4	,,	7 days	Do.	Do.	None

General symptoms.	Duration.	Organisms found.	Virulence of the K.-L. bacilli.	Family surroundings of patient.
Languor, pallor, &c.	12 weeks	Pure short K.-L.	2·0 c.c. in 2 days	Father well; mother sore throat sister sore nose. Three othe families in house remained wel
None	4 weeks	Short K.-L. mixed	Died out	All well.
None	3 weeks	Large K.-L. mixed	2·0 c.c. in 2 days	Mother and one sister = well; on sister = K.-L.
Pale and languid	5 weeks	Large K.-L. pure	Do.	Father = well; mother and sister = sore throats. Two families wit nine children = well.
Slight debility	18 weeks	K.-L. and cocci	Died out	All well.
Feverish at first	7 weeks	Do.	Do.	Two adults and five children = wel
None	+ 4 wks.	Pure large K.-L.	2·0 c.c. in 3 days	All well.
Very slight	3 weeks	Do.	2·0 c.c. in 2 days	All well.
Slight	5 weeks	Large K.-L. mixed	Died out	All well.
Do.	5 weeks	Large K.-L. pure ?	Do.	All well.
Headache, pallor, and weakness	4 weeks	Large K.-L. mixed	2·0 c.c. in 2 days	Father, mother, and two childre = well.
None; at school all through	7 weeks	Short K.-L. mixed	Not tested ⎫	Sisters. One other sister, fathe and mother = well. Two othe families, all well.
None	3 weeks	Do.	Not tested ⎭	
Slight	6 weeks	Large K.-L. mixed	Died out	Another child sore nose.
School all through	6 weeks	Pure large K.-L.	0·5 c.c. in 2 days	Three families, all well.
None (supposed foreign body in nose)	3 weeks	Do.	0·5 c.c. in 1 day ⎫	
None	3 weeks	Do.	0·5 c.c. in 3 days ⎭	Sisters. Father and mother = wel
Poorly, but not really ill	5 weeks	Large K.-L. mixed	Died out in spite of agar plates and nearly pure culture	—
Do.	5 weeks	Do.	0·5 c.c. in 3 days	Four adults well; five childre one with sore throat.
Very slight	5 weeks	Large K.-L. pure	0·5 c.c. in 2 days	One child with sore nose; oth children and mother = well father said to have diphtheria.

CONGENITAL HYPERTROPHIC STENOSIS
OF THE PYLORUS

BY

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THE condition illustrated by the cases described in this paper is one in which the pylorus of the infant is found *post mortem* to be represented by a well-defined, cylindrical thick-walled tube, with its lumen exceedingly small or completely occluded.

There are other malformations of the pylorus which may cause obstruction during infancy or in later life. Thus there may be complete atresia, as in the cases recorded by Neale (1) and Lesshaft (2) ; or there may be a simple narrowing of the orifice, which may or may not be surrounded by a ring-like thickening. Henschel's (3) cases, referred to below, and those recorded by Finkelstein (4) from Heubner's clinic appear to be instances of simple stricture. Other causes of obstruction, such as polypi of the mucous membrane, recorded by Goldenhorn and Kolatchewski (5), and carcinoma of the pylorus (6) in a child aged five weeks, may be regarded as pathological curiosities.

The kind of stenosis under consideration is more common, although comparatively little attention has been paid to it, and is of considerable importance. Stress has been laid on congenital narrowing of the pylorus as an important factor in the pathology of the dilatation of the stomach in adults. It is by no means improbable that the condition of pyloric stenosis in adults, described by Habershon (7) in 1862 as " fibroid degeneration of the pylorus," and by Lebert (8) in 1878 as " hypertrophic pyloric stenosis," is due to the progressive development of a similar condition in infancy.

In his Inaugural Address in 1879, Landerer (9) described cases of stenosis and hypertrophy of the pylorus, occurring in adults and older children, which he regarded as congenital in origin. One of his most typical cases was that of a man aged forty-five, who had suffered since infancy from dyspepsia, a tendency to vomiting, and troublesome swelling of the abdomen. Death resulted from general atrophy due to impaired nutrition, and *post mortem* simple stricture of the pylorus and a dilated stomach were found. Prof. Rud. Maier (10) at Freiburg in 1885, under whose auspices Landerer's paper had appeared, dealt with the subject in an exhaustive manner, and detailed the symptoms of thirty-one cases as persistent vomiting and dilatation of the stomach. The two youngest of his patients were twelve and sixteen years of age respectively. He considered that a degree of congenital stenosis is a frequent, if not the most frequent, though unrecognised cause of this condition. Demme (11) in 1881 recorded a case of dilatation of the stomach in a boy aged six and a half years, who had suffered from vomiting and constipation since birth. He ascribed it to a contracted cicatricial ulcer, but more probably it was due to a certain amount of congenital stenosis. Tilger (12) in 1893 described similar cases, almost all in adults.

So far it does not appear at all clear that the hypertrophy of the pylorus in adults is secondary to the stenosis rather than the stenosis secondary to the hypertrophy.   I

am very much inclined to accept the latter view. To prove the connection between the hypertrophic pyloric stenosis in adults and a similar condition in infancy, it is necessary to find evidence of such a condition in early life.

The first record of the affection in infancy is a report by Williamson (13) in 1841 on "A case of scirrhus of the stomach, probably congenital." The specimen was exhibited at a meeting of the Anatomical Society of Edinburgh, and was regarded by the members as scirrhus of the pyloric extremity of the stomach. The details of the case correspond with those of more recent date.

CASE 1.—Male, apparently healthy at birth. Vomiting came on in a few days, and death took place in five weeks from exhaustion. *Post mortem* the pylorus was found hard and indurated, the orifice barely admitting a small silver probe. The mucous membrane and the submucous cellular tissue were thickened, the latter so much so as to form almost the whole wall.

Williamson regarded the condition as a "peculiar hypertrophy or modification of the cellular tissue." He was not aware of any other case.

Dawosky (14) in 1842 reported a case which came under his observation in the previous year.

CASE 2.—Child aged nine weeks when first seen, apparently healthy at birth. It was satisfactorily breast-fed for four weeks, and then vomiting occurred two or three times daily. The vomit consisted of curdled milk and mucus. The stools were small. Death took place in the tenth week from marasmus, preceded for some days by convulsions. *Post mortem* a large hard pyloric tumour the size of a "nut;" the lumen would hardly admit a probe.

He regarded it as a hypertrophy with induration of the submucous tissue. After this date no attention appears to have been attracted to this affection until Hirchsprung (15) of Copenhagen in 1888 recorded two cases.

CASE 3.—Thora J—, youngest of three girls, born December 27th, 1880, at term, and apparently healthy. She was breast-fed and well until ten days after birth. Severe vomiting then came on, and she died on the thirtieth day. Constipation was a very prominent symptom. *Post mortem* the œsophagus was found dilated and its walls thickened, the wall of the stomach somewhat thickened throughout, and the pylorus represented by a firm cylindrical thickening, 2½ cm. long, through the lumen of which only a moderately thick sound could be passed. All the layers, particularly the muscular, were hypertrophied, and the mucous membrane was thrown into six projecting parallel folds.

CASE 4.—Nathalie O—, born April 13th, 1886, came under treatment on July 9th of the same year. At two weeks of age she was put out to nurse and fed in various ways, but her digestion was always bad, and she vomited repeatedly. At first she had diarrhœa. Death took place at six months from tuberculosis. The pylorus formed a thick cylindrical swelling, 3 cm. long, due to hypertrophy of the muscular tissue. The stomach was a little dilated, but not the œsophagus. Vomiting was a pronounced symptom during the whole of the child's life, but the obstruction at the pylorus was not complete, an ordinary pencil could be passed through it.

In 1889 W. K. Peden (16) showed a specimen of this condition at a meeting of the Glasgow Pathological and Clinical Society.

CASE 5.—A well-developed baby at birth. The three previous children were all great vomiters. Vomiting began three days after birth; it was persistent and characteristic; a large drink was immediately rejected, small ones were retained until a certain quantity had collected. No tumour could be felt. The tongue remained clean, the breath sweet, and the bowels confined, but not markedly so. The child died at the age of

three months. *Post mortem* the pylorus formed a sausage-shaped thickening, with a lumen 5 mm. in diameter, further narrowed by two longitudinal crest-like projections extending along the whole length of the hypertrophied portion.

In 1891 G. Newton Pitt (17) showed a similar specimen at a meeting of the Pathological Society of London.

CASE 6.—The child died at the age of seven weeks, having suffered from vomiting and constipation for three weeks. The pylorus was an inch long, and formed of hypertrophied muscle. The wall of the stomach was also thickened.

Henschel (18) in 1891 recorded four allied cases, one of which recovered.

(i) Male aged 16 months ; had suffered with chronic gastric catarrh, habitual constipation, and temporary diarrhœa. The stomach was dilated and the pylorus thickened, but its lumen would admit the little finger.

The next three cases were all in one family.

(ii) Female aged 19 months. The parents had long suffered from gastric troubles. The child vomited everything for the first six months, and was constipated. It improved for a time, and died at two years from exhaustion and excessive vomiting. The stomach was found dilated and the pylorus thickened, the lumen admitting a thick lead pencil.

(iii) A sister died at 7 months with similar symptoms. No autopsy.

(iv) A brother suffered with vomiting, constipation, and tympanites. He got better, and was seen when two and a half years of age. A fourth child of this family died of tuberculosis, and had abnormalities of the pylorus.

None of these cases of Henschel's presented the typical thickening of the pylorus which I am considering. They are rather instances of partial narrowing of the pyloric orifice, and are valuable as indicating the liability

of this orifice to congenital or infantile pathological changes.

Dr. John Thomson (19), of Edinburgh, published two cases in 1896 and a third in 1897. In the second paper he gave a valuable bibliography. These two papers have been very kindly placed at my disposal by the writer, and I take this opportunity of gratefully acknowledging my indebtedness to him.

CASE 7.—Male, apparently healthy at birth and for ten days after. Then continual vomiting until death on the twenty-eighth day from emaciation and exhaustion. *Post mortem* the œsophagus and stomach were found dilated, the mucous membrane of the latter congested. The pylorus was thickened and elongated, its walls one sixth of an inch thick, and the mucous membrane thrown into folds. The lumen would admit probe-pointed scissors. The condition was due to hypertrophy of the circular muscular fibres.

CASE 8.—Male, apparently healthy at birth and for four weeks after. Then vomiting occurred almost every time he took the breast, and continued, in spite of weaning and various methods of feeding, until death, preceded by fits, in the tenth week. The bowels were moved regularly during the first seven weeks of life, but after that there was obstinate constipation. The *post-mortem* appearances were similar to those of the last case.

CASE 9.—Female, apparently healthy at birth and breast-fed for a week. Diluted cow's milk was then given, and vomiting began at once and continued, occurring immediately if more than two ounces of food were given at a feed. Gradual emaciation. A small hard moveable tumour was sometimes felt in the epigastrium. Death resulted on the forty-seventh day, preceded by convulsions for five days. *Post mortem* the same appearances were

found as in the other two cases, with the addition that the submucous connective tissue was much thickened.

Chr. Gran (20), of Christiania, in 1896 described three cases.

(v) Female, died at four months, after vomiting and diarrhœa for a month. *Post mortem* the stomach was found much dilated and the pyloric orifice constricted.

CASE 10.—An illegitimate female, first child of a healthy mother. Vomiting and diarrhœa began at two months. After that there were frequent vomiting and an inclination to constipation. Death took place in the fifth month from exhaustion due to diarrhœa and vomiting. The digestion of milk was found to be more imperfect than that of farinaceous fluids. The pylorus was thickened and contracted, its lumen measuring 3 mm. in diameter. The thickening was due to hypertrophy of the muscular fibres, especially the circular ones.

CASE 11.—Male child, died in the fourth month, after attacks of vomiting and diarrhœa. The stomach was found very dilated. The pylorus was thick, the muscle extremely strong and thick, and the lumen was constricted.

H. Finkelstein (21) published a paper in 1896 in which he refers to the cases of Hirschsprung, Henschel, and Gran, and records another.

CASE 12.—Female, brought up on cow's milk from birth, had started vomiting within a few days. During the third month the vomiting became more frequent, and there was much wasting and constipation. A tumour could be felt, about 2 cm. above and somewhat to the right of the umbilicus, when the child was three months old, and death took place a few days later. *Post mortem* the characteristic cylindrical thickening of the pylorus, forming a muscular " intermediate piece ; " much swelling of the mucous membrane, almost occluding the lumen.

He also refers to three cases from Heubner's clinic, which presented symptoms of this condition in early life. The diagnosis could not be verified, as the patients recovered.

De Bruyn Kops (22) in 1896 reported one case.

CASE 13.—Male, well nourished at birth, began vomiting some hours after, and died at the end of three weeks of emaciation and anasarca. The pylorus was of the size of a "marble," and of cartilaginous hardness; it was much hypertrophied and markedly stenotic.

F. Schwyzer (23) in 1896 reported one case.

CASE 14.—Female, weighing over eight pounds at birth, thrived well on the breast for two weeks. Then occasional vomiting and repeated attacks of diarrhœa. Death in the tenth week. The pyloric tumour was 2·4 cm. long, and the lumen admitted a small probe with difficulty.

Ashby (24) in 1897 published two cases.

CASE 15.—An infant started vomiting after being quite healthy for a week. Gradual emaciation, convulsions, and death. The characteristic hypertrophy and stenosis of the pylorus were found *post mortem*.

(vi) An infant, operated on successfully on the third day of life for imperforate anus, died on the fifth day from continued vomiting. Much pyloric stenosis and some hypertrophy of the surrounding muscle were found *post mortem*. The characteristic sausage-shaped tumour is not described as being present.

CASE 16.—There is a specimen of the condition in the St. Bartholomew's Hospital (25) Museum (No. 1907a), presented by J. Raglan Thomas, M.D. It was obtained from an infant who began vomiting at the age of three weeks, and died from inanition at seven weeks. The child was healthy at birth. The pylorus presents the usual condition. The stomach wall is thickened and the

œsophagus dilated. The mucous membrane of the stomach and pylorus is congested and swollen. Microscopically an overgrowth of unstriped muscular tissue. The thickening terminates abruptly on the duodenal side, and gradually tapers off towards the cardiac side of the stomach.

CASES 17 AND 18.—W. Soltau Fenwick (26), in his work on the " Disorders of Digestion in Infancy and Childhood," 1897, refers briefly to two cases which came under his notice at the Evelina Hospital. I can find no specimen of this condition in the museum of the Royal College of Surgeons.

The details of the two cases under my care are as follows :

CASE 19.—Henry Kendall T— attended as an out-patient at the Belgrave Hospital for Children on April 20th, 1897, aged 11 weeks.

The father and mother are alive and healthy. The mother had two children by her first husband, the second of which died. Patient is the fifth child by the second husband, the other four being alive and healthy.

The child was apparently healthy at birth and up to the age of five weeks, when it began to waste rapidly. The mother's account is somewhat vague and unreliable, but as far as can be ascertained vomiting began at the age of three weeks. It is insured, and all the other children also.

He was brought up on the breast for three weeks, and then on the breast and Mellin's food. He vomited everything, and was then tried on condensed milk and barley water; this also was vomited. The bowels have been obstinately constipated since birth, and only opened by enemata.

Physical examination.—Much wasting ; head markedly dolichocephalic, fontanelles closed ; inguinal hernia on the right side ; tongue coated with a white fur, breath

sour. Vomiting occurs after feeding, the vomit being sour. The bowels costive.

April 20th.—Profuse nasal catarrh. The mother gives the breast every two hours, and has enough milk. Vomiting has taken place only once a day. Much flatulence. The bowels acted naturally yesterday, the stool being a good colour.

May 3rd.—The child cries more and is in pain. He vomits twice a day, bringing up a "quantity of thick material." The bowels have acted on alternate mornings as a result of half a grain of grey powder twice a day since his last attendance. The wasting has increased. He was admitted. Weight 7 lbs. 10 oz.

Course.—He gradually became weaker. He vomited every kind of food at varying intervals. Peptonised milk in small quantities was kept down for a couple of days, but he lost weight on it. The bowels acted sometimes without the aid of medicine, the stools being small and often greenish. The temperature ranged between 96° F. and normal, being generally at the lower level. After May 8th the temperature was constantly below 97° F., and the vomiting became more frequent. Death resulted from exhaustion on May 12th. There was a loss of 6 oz. during the first three days after admission, but after that the weight remained practically stationary.

Post-mortem.—Great emaciation. Head large and heavy. All the visceral organs appeared healthy with the exception of the stomach and pylorus. The pylorus was represented by a firm thick sausage-shaped swelling, an inch in length, distinctly limited above and below. Looked at from the duodenal side it has a remarkable resemblance to the os uteri. The stomach was much dilated, and on squeezing it firmly, pressing the contents towards the pylorus, a small quantity could be forced through into the duodenum. A small amount of food was found in the intestines.

On opening the stomach the contents were found to consist of partially digested food and mucus. The

pyloric orifice was very small, and would only admit a probe. On opening the pylorus by an incision prolonged from the lesser curvature of the stomach the wall was found very thick, about three-eighths of an inch ; the mucous membrane was thickened, especially towards the duodenal end, where it formed almost a definite capsule, thinning off on the cardiac side into the mucous membrane of the stomach. On the side corresponding with the continuation of the greater curvature of the stomach the mucous membrane was thrown into a thick ridge-like fold, continuous throughout the whole length of the pyloric tumour. The resistance on section of the wall was almost as firm as, and somewhat similar to that experienced in cutting through a scirrhous carcinoma of the pylorus. To the naked eye the main cause of the thickening is a remarkable increase in the circular muscular fibres, this thickening being most pronounced on the duodenal side and tapering off towards the cardiac side, but nevertheless ending fairly abruptly on that side. It is perfectly easy to see the limit of the hypertrophy. It is very evident that the thickening of the mucous membrane is an important factor in the production of a more or less complete obstruction.

Microscopical examination confirms the above observations. The mucous and submucous coats are markedly thicker than normal, but the great factor in the production of the sausage-shaped swelling is an extensive hypertrophy of the circular muscular fibres.

A further abnormality was found in the shape of a large cavity in the right hemisphere of the brain, somewhat irregular, lined with smooth walls, and containing clear fluid and a few flakes of lymph. This cavity did not appear to communicate with the ventricle, though at one time it may have done so.

It is curious, when we consider the rarity of this condition, that the second case came under my notice a few days after the death of the first child. For the clinical

notes and the details of the *post-mortem* I am indebted to my house physician, Mr. G. S. Haynes.

CASE 20.—John I— at the age of seven weeks was brought to the hospital on May 17th, 1897, for wasting. He was brought by a " friend," paid to look after the child by the mother, who was out at work as an ironer. This friend stated that the child was " a fine baby born." He had been wasting for a week, and vomited invariably after feeding. He was fed irregularly on the breast and condensed milk. The stools were green and offensive.

On May 19th the child was brought by the grandmother, who could give no information about it.

On May 20th at 6 a.m. he died " in a fit." The instructions as to feeding had not been carried out. The coroner, having been notified, ordered a *post-mortem* examination.

According to subsequent information obtained from the mother, the child was the youngest of four, of whom one other is dead. The father suffers from rheumatic gout, and is in the infirmary. The baby was not weighed at birth, but was a fairly good size. During the first week of life he vomited frequently, although only breast-fed. At the age of three weeks he was taken to the Western Dispensary and treated there for three weeks. As he did not improve he was taken to the Westminster Hospital, where cow's milk and barley water were prescribed for him. When brought to the Belgrave Hospital he was cold, very emaciated, but had a fair pulse.

Post-mortem.—Extreme emaciation. On opening the abdomen the distended stomach was seen extending obliquely from the left hypochondrium to the right of the umbilicus. The splenic end was in contact with and adherent to the spleen, which was somewhat large. The stomach contained about four ounces of darkish grey fluid, rather slimy from the presence of an excess of mucus, and blackish particles of matter. Its mucous membrane was pale and swollen. The pylorus presented the same

characteristics as in the other case, though not quite so marked. It formed an elongated sausage-shaped tumour about an inch long, and its lumen was impervious. The small and large intestines were absolutely empty. The appearances on section to the naked eye and on microscopical examination were the same as in the previous case. No other abnormality was present.

Summary.

Here are no less than twenty undoubted cases in which the pylorus presented a remarkable condition of muscular hypertrophy and stenosis. There are six cases (Nos. 1—6) which presented conditions of stenosis but not the peculiar hypertrophy, and four others which presented fairly characteristic symptoms and recovered. The cases have been collected from various parts of the world, and it is noteworthy that all but two have been recorded during the last ten years, and no less than eleven (including Fenwick's cases) during the last two years. It is evident that the condition is not so extremely rare as might be supposed. In all probability many more instances of it would be found if *post-mortem* examinations were invariably made on infants dying from vomiting and marasmus. It is distinctly the cause of that symptomatic disease " marasmus " in some cases.

General Description.

The most definite clinical symptom of this affection consists in constant vomiting without apparent cause, especially in the absence of any sign of gastritis or enteritis. The next important symptom is more or less constipation. Gradually the child emaciates, and death results from exhaustion consequent on general failure of nutrition, and is sometimes preceded by convulsions.

Family history.—Nothing has been noted in the parents as likely to account for the condition. There appears to

be no indication whatever of any connection with inherited syphilis. As a general rule the other children of the family are healthy. Possibly there may be a predisposition in certain families. Thus in Peden's case it was noted that the three previous children were all great vomiters, and three of Henschel's allied cases occurred in the same family. In the two cases under my own care there was no indication in the family history of such a predisposition or of an inherited ætiological factor.

Sex.—It is not to be expected that one sex should be more liable than another. About half the patients are male and half are female.

Condition of the child at birth.—In every case it has been noted that the infant was apparently healthy at birth, or, to use a common expression, was " a fine baby born." There is no indication at this period of the presence of the pyloric affection.

Date of onset.—This varies considerably. The first symptom may be noted a few hours after birth (Case 13), or may not be noted for a month or more. As a general rule it is rare for the vomiting not to commence during the first month of life. No doubt the actual date depends upon the degree of stenosis present.

Mode of onset.—This invariably takes the form of vomiting. Until the child is sick there is no indication that it is not in perfect health, unless it suffers from a certain amount of flatulent dyspepsia and constipation. Both the latter symptoms usually follow the vomiting in point of time.

Characteristic symptoms and physical signs.—1. *Vomiting.*—At first this may only occur at fairly long intervals,—once a day, for instance, a considerable quantity of food being pumped up when the stomach has become over-distended. Gradually it becomes more and more frequent, several times a day. It is persistent and very characteristic, A large drink is invariably rejected almost immediately. but small meals may be retained a considerable time, after which several are vomited at once. In the first of my

cases on one occasion there was no vomiting for forty.
eight hours, but the amount of food taken by the child
was not enough for the maintenance of nutrition; the
child lost weight, and its temperature was constantly sub-
normal. In the later stages vomiting occurs on the
administration of the smallest quantities of food.

At first the vomiting may be mistaken for the simple
regurgitation of food so common in infants. Later on
such an error cannot be made if the child is under skilled
observation. The act is so much more forcible, more
like the vomiting of older children, and it apparently
causes a very definite amount of pain. Nevertheless the
infant is most comfortable when the stomach is empty.

The nature of the food does not usually influence the
vomiting. It depends mainly on the amount. To a certain
extent soluble foods, such as saccharine solutions, and
pre-digested foods such as peptonised milk, are kept down
better than ordinary milk. Thus in my first case I found
that peptonised milk was retained best, at any rate for a
period of two days. Gran found in one case (No. 10) that
thin farinaceous fluids were retained better, and were more
perfectly digested than milk, probably for the simple
reason that the latter is a much more complicated fluid.
The vomited matter consists at first entirely of the food
given, more or less changed by the action of the gastric
juice. It is consequently composed of sour coagulated
milk, this being the common diet in infancy. Frequently,
especially in the later stages of the illness, there is a
considerable quantity of mucus mixed with it.

There is no bile in the vomit. This is a very important
fact, for it indicates that the seat of obstruction is above
the entrance of the common bile-duct into the duodenum.

If the stomach be washed out and a test feed inserted,
it will be found possible to recover the whole or a greater
part of the feed in three or four hours' time. Gran made
some interesting observations (loc. cit.) under such cir-
cumstances. This is a valuable test of the presence of

pyloric obstruction, for it is generally accepted that the stomach of infants is emptied in about two hours' time.

2. *The bowels.*—Constipation, though a common symptom, is not as invariably present as would be expected. In the early stages, and even in later stages when the obstruction is not complete, there may at times be diarrhœa. This is due to irritation from decomposing food retained in the stomach, the irritating products passing on into the intestines. Generally the stools are small, and present no obvious alteration. In some cases constipation is a very marked symptom, the bowels not being open for eight days (Case 3), or only open by means of enemata (Case 19). Even during the last fortnight of life there may be diarrhœa, but, as a rule, there is obstinate constipation.

3. *The tongue.*—The character of the tongue varies according to the absence or presence of secondary gastric catarrh. It may be clean throughout the illness (*e. g.* Case 5), and at the same time the breath may be quite sweet. Or the breath may be sour and the tongue coated with a thin white fur.

4. *The abdomen.*—On inspection there are no signs of intestinal obstruction. The abdomen is flat and thin. There may be a distinct swelling in the epigastrium and the usual signs of a dilated stomach. On the other hand, it must be remembered that vomiting takes place with such great ease in infants that there may be no very definite increase in the size of the stomach. A dilated stomach may also occur in conditions unconnected with pyloric obstruction.

It may be possible to demonstrate the presence of a tumour in the region of the pylorus. Finkelstein demonstrated an enlarged hard pylorus (Case 12), and Thomson found a hard moveable tumour in the epigastric region (Case 9) during life.

5. *General condition.*—There are all the signs of starvation. The child is very wasted, and continues wasting. The surface of the body is cold, and the tem-

perature is subnormal. The pulse is frequent, small, and weak. The child lies in a condition of lethargy, with half-open eyes, and takes no interest in its surroundings. It occasionally cries from pain, and rouses a little to vomit. Food is not taken with avidity, and is often refused.

Morbid Anatomy.

There is great emaciation. All the organs of the body are free from the signs of disease with the exception of the alimentary tract. In one case (No. 4) tuberculosis of the lung and secondary dissemination were present and caused death, the stenosis not being at all extreme although the pyloric hypertrophy was well marked.

The *oesophagus* may or may not be dilated, dilatation depending on the degree and duration of the obstruction.

The *stomach* generally presents more or less dilatation, sometimes extreme, in which case its walls are thinned. The contents consist of undigested food and mucus, and occasionally small particles of blood. The mucous membrane is normal unless there has been co-existent gastric catarrh, in which case the ordinary signs of this condition will be found. In some infants the muscular walls of the pyloric end of the stomach have been found somewhat hypertrophied. I could not satisfy myself that such hypertrophy was present in my cases.

The *pylorus* is represented by a very definite tumour, which has been described as an "intermediate piece," a "cylindrical swelling," and as a "sausage-shaped tumour." It varies a little in size, but may be taken roughly as about the size of the last joint of the little finger, and about an inch long. On external examination with the fingers the limits of the tumour can be definitely fixed both on the duodenal and the gastric side; to the touch it ends more abruptly on the duodenal side, but there is also a very distinct limit to it on the gastric side. It is very firm and hard. If the duodenum is laid open and the pylorus looked at from that aspect, it

resembles the os uteri in having a small central orifice, apparently occluded, and a thick, smooth, ring-like surrounding wall, and it projects into the duodenum in much the same manner as the os uteri projects into the vagina. The appearance from the stomach side is rather funnel-shaped, and the occlusion of the lumen by the puckered mucous membrane is more distinct.

To the naked eye the occlusion of the lumen is complete in some cases, and it is not possible to squeeze the fluid contents of the stomach through it. Even in these instances a fine probe can be passed through the whole length of the canal. This occlusion of the lumen is due to the contraction of the hypertrophied muscular tissue forming its wall, and is assisted by the thickening of the mucous membrane, which is thrown into one or more longitudinal ridge-like folds extending the whole length of the thickening.

· On longitudinal section it can be seen very clearly by the naked eye that the tumour is simply a thick-walled tube, and it is evident that the main cause of the thickness of the wall is the hypertrophy of the muscular fibres forming the middle coat. This hypertrophy is most marked and ends most abruptly on the duodenal side, forming a rounded swelling, and it gradually diminishes though still ending fairly abruptly on the gastric side. The mucous membrane and submucosa appeared definitely thickened in my two cases. The single ridge-like fold in the specimen from Case 19 is extraordinarily marked. On looking at the cut surface, the mucous membrane is seen to form a very definite capsule, especially thick over the intra-duodenal portion, and thinning off gradually into the mucous membrane of the stomach.

Microscopical examination confirms the above observations with the exception that it shows the mucous membrane is not thickened, and that the apparent thickening is only due to the contraction of the walls of the tube. The hypertrophy of the circular muscular

fibres is very marked, and there is a moderate increase in the amount of intermuscular connective tissue. The external longitudinal muscular layer is a little thicker than normal. There is no change in the serous covering and no sign of inflammatory action.

These observations practically agree with those of previous observers. Finkelstein, however, states that the thickening in his case (No. 12) was due to hyper-trophy of the longitudinal muscular fibres, and gives a diagrammatic representation of the specimen in his paper I am inclined to think that the appearance is due to the section having been cut obliquely.

Pathology.—The condition is definitely one of hyper-trophy of the circular muscular fibres which are normally present in the pylorus. In considering the origin of the hypertrophy we must bear in mind the following facts.

1. It is a pre-natal condition, for the extent of the hypertrophy is such as could not have occurred during the few weeks of extra-uterine life. In several cases the symptoms were observed during the first week of life.

2. There is plenty of time for the hypertrophy to occur before birth, for the first indication of a pylorus is found during the third month. It is formed from the fore-gut and the circular muscular fibres are derived from the mesoblast.

3. Normally the pylorus of an infant is represented by a small circular orifice, surrounded by a slight increase in the circular muscular fibres belonging to the general system of circular muscular fibres of the alimentary canal Externally it can be felt as a slightly thickened ring. I is evident that in the condition under consideration the amount of hypertrophy is relatively enormous.

The general explanation of hypertrophy is increased work. In the present instance it is difficult to find th source of an increase in work. Thomson, in his second paper, puts forward the theory that " the essential lesion is not a muscular but a nervous one—a functional disorder of the nerves of the stomach and pylorus leading to ill

co-ordinated and therefore antagonistic action of their muscular development." He regards the affection as a " congenital gastric spasm," or " congenital pyloric spasm."

Another theory which might be put forward is that it is due to a simple redundancy of fœtal growth. Nature, in her extreme anxiety to provide an efficient pyloric sphincter, has over-exerted herself, and produced too great a quantity of muscular tissue. It is useless to discuss theories in the present state of our knowledge of the affection, and I prefer to retain a name which indicates the anatomical condition, and thus at once brings to mind the symptoms likely to arise from that condition.

Diagnosis and prognosis.—The *diagnosis* depends on the characteristic vomiting, the absence of bile from the vomit, the presence of constipation, a history of the occurrence and persistence of the symptoms without definite cause and in spite of treatment, and finally in the presence of a cylindrical moveable tumour in the region of the pylorus.

The affection is especially liable to be confused with the simple regurgitation of food in infants and with simple gastric catarrh. The character of the vomiting distinguishes it from the former, and the presence of a clean tongue and sweet breath will eliminate the latter. The presence of a tumour is almost diagnostic, for other forms of tumour in this region at an early age are extremely rare, *e. g.* an enlarged gland is rarely sufficiently definite, and if present would hardly be likely to cause such symptoms.

The *prognosis* is necessarily bad, especially if the tumour is large enough to be felt. Cases of recovery from symptoms characteristic of the disease have been referred to, but the diagnosis must necessarily remain doubtful. The duration of life depends upon the severity of the stenosis. The shortest period recorded as elapsing between the onset of the vomiting and a fatal termination is seventeen days. The longest is six months (Case 4),

but here the cause of death was tuberculosis. Fou
months of life is about the average for infants thu
affected. In the slighter instances of this affection it i
reasonable to suppose that the symptoms may not be o
much moment, and may be limited to dyspepsia with ar
occasional attack of vomiting. It is quite possible tha
such cases do exist, and that they terminate in later lif
from dilatation of the stomach, hypertrophic stenosis o
the pylorus being found post mortem.

Treatment.—If the tumour can be felt, and an accurat
diagnosis thus arrived at, the most rational treatmen
consists in operative measures. Three courses would b
open to the surgeon. The pylorus might be dilated, o
it might be excised, or a communication might be mad
between the stomach and the small intestine. The las
method would probably be the most satisfactory.

Apart from surgical treatment, only palliative measure
are available. Drugs are useless to relieve the vomiting
Lavage of the stomach is of temporary benefit by washin
out mucus and decomposing food, thus allowing the chil
a better chance of absorbing some of its next meal. Smal
quantities of food should be given at a time, and the foo
should be pre-digested and of simple composition. No
much can be expected from these measures, for in infant
the stomach has not a capacity for the digestion o
absorption of more than a small amount. Schwyzer kep
his patient alive for a fortnight by rectal feeding.

I cannot claim to have added new facts to our know
ledge of this uncommon affection, nor to have devise
new methods of diagnosis or treatment. As far as I an
aware, no paper has been read on this subject before
medical society in London, and I have taken the oppor
tunity afforded me by the two cases which came unde
my notice of bringing the subject before you, with th
object of eliciting the experience and the views of mor
competent authorities on this very curious and fatal con
dition.

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A DISTINCT VARIETY

OF

HIP-JOINT DISEASE IN CHILDREN AND YOUNG PERSONS

BY

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THERE is slight risk of the statement being traversed that disease of the hip-joint, as met with in young people, is usually of tuberculous origin. To make the statement more absolute, let there be taken away those instances of traumatic synovitis or arthritis which clear up after a week's rest, and those cases of syphilitic and osteo-arthritic inflammation, which, after all, are very rarely met with. Then, surely, most of the remaining cases are of tuberculous origin.

The ground being thus cleared, there remains a very important group of cases of hip-joint disease which should be at once recognised, and picked out from amongst the crowding tuberculous cases by which they have been too

apt to be overshadowed and obscured, and with which they have been not infrequently confused.

The cases to which by this paper I desire to direct attention are those in which septic disease at the upper end of the diaphysis of the femur spreads into the synovial cavity, and rapidly involves the hip-joint in an acute and devastating suppuration. In the surgery of childhood there is no disease more sudden in its onset and disastrous in its effects than acute arthritis of the hip, started by septic osteomyelitis.

Before going further, I may perhaps be permitted to make some remarks upon the anatomy of the parts concerned.
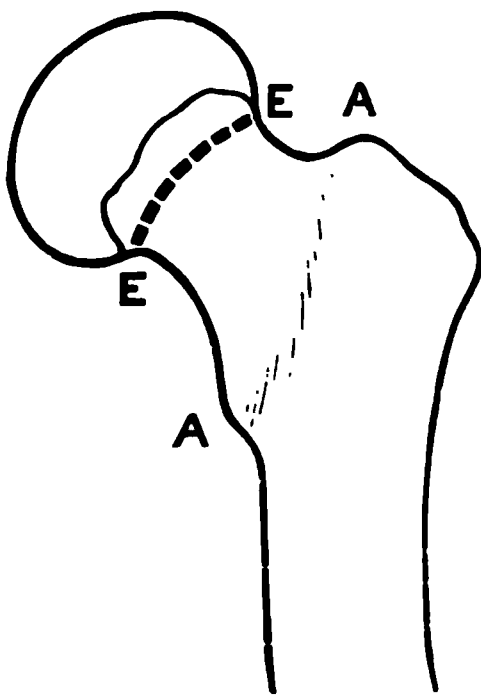
The hip-joint differs from other joints in this, that the end of a diaphysis enters into its formation. The upper end of the growing femur is represented in this sketch, which shows at A A, the attachment of the capsular ligament to the anterior intertrochanteric line, and at E E the junction of the diaphysis and epiphysis. The whole of the neck of the femur belongs to the diaphysis, and is placed within the capsule. The epiphysis is separated from the upper part of the neck by a plane of temporary cartilage, and on the diaphysial side of this cartilage additional cells are constantly being laid down and as constantly being converted into bone. This ossifying cartilage is of great physiological activity and structural delicacy, and is especially prone to be attacked by

the micro-organisms of septic osteomyelitis, as well as by the bacilli of tuberculosis.   In a considerable proportion of the cases of septic osteomyelitis, the invasion of the germs is invited by some injury to the limb, which, setting up an ordinary inflammation, has thereby lowered the vitality of the tissue and rendered it an easy prey to septic organisms.

The growth of these germs suddenly adds great intensity to the inflammation, and gives rise to a series of local and constitutional disturbances which generally serve to distinguish it.   As the head of the femur ossifies on to the shaft about the nineteenth year, the subject of the disease is always under twenty.

There are other causes besides injury which predispose to the successful cultivation of the septic micro-organisms in this vulnerable tissue,—any condition, indeed, which lowers the vitality of the tissue, and thereby renders it less capable of resisting the invasion of the staphylococci. Thus scarlet fever, measles, enteric fever, or influenza may be the precursor of the attack.

1 am inclined to think that in a considerable proportion of those cases in which the surgeon opens the hip-joint and finds the solid head of the femur detached from the neck, and scarcely affected by carious disintegration, the cause of the separation has been septic inflammation just below the junction-cartilage.   The fact of the detached epiphysis being sound, and its contour even, is proof that the separation occurred early in the course of the disease, and at a time when the joint was unoccupied by granulation tissue.   When the epiphysis becomes detached in the course of tuberculous disease, not only its epiphysial aspect gives evidence of rarefactive inflammation, but even its convex surface appears soft, worm-eaten, and irregular.

All cases of septic inflammation of the upper end of the femoral diaphysis are not equally acute and urgent, nor do they all entail suppurative arthritis.   In some instances the thrombosis is limited to quite a small area of the para-epiphysial tissue, and the resulting sequestrum may

eventually be removeable without opening the joint, by tunnelling through the great trochanter. Interesting as these cases are, however, they have only a collateral bearing upon the subject of this paper.

Symptoms.—The two great features of the symptoms of this form of hip-joint disease are their acuteness and the suddenness of their appearance. In these respects the disease differs widely from tuberculous arthritis. For though each may come on after an illness or a hurt, it is the nature of the tuberculous disease to make its appearance slowly, and thus a faulty position of the thigh, a wasting of the muscles, and a limping gait may have existed for some while before the lesion is actually declared. Not so, however, with the septic disease : the child is attacked suddenly, and in the course of a few days the symptoms may have attained alarming intensity. In the case of tuberculous disease there is often a history of a long smouldering of equivocal symptoms before the flame breaks forth, but in the septic arthritis the blaze is almost instantaneous. The child looks extremely ill, and though its face may at times be flushed it may be extremely pale, and the expression anxious. There will probably be a history of sleeplessness, and most likely of delirium.

As a rule, the temperature is high—up to 104° F. (40° C.), may be. But if, by chance, the first thermometric observation is taken when the disease has existed for some while, it may show no elevation but actually a depression. The explanation of this is that the dose of toxic material elaborated by the growth of the micro-organisms may be so intense that the heat-producing centre, like every other area, is profoundly affected ; this general depression may end in a fatal collapse.

As regards the local symptoms, the child dreads the least disturbance of the limb. Probably he cries out or screams when any attempt is made at examination, pointing to his hip and thigh when asked where the pain is. On his turning in bed, or on an attack of muscular

spasm coming on at night, the pain is intensified, and he starts up with a scream.

The limb is generally found in a position of flexion and adduction ; there is probably some swelling of the thigh, especially below the middle of Poupart's ligament, and oh gently squeezing the articular end of the femur between the fingers placed at the front and at the back of the joint, some fulness may be detected, and it is evident that the pressure causes intense pain. Pressure upon the outer side of the trochanter also causes distress.

Error of diagnosis may easily be made, the case being probably mistaken for one of acute rheumatic affection. Or it may be regarded and dealt with as one of tuberculous inflammation with unusually acute symptoms. Or, without recognising its exact pathological nature, the practitioner may write it down as a case of acute hip-joint disease, which truly it is.

Some of the subjects of this form of hip-joint disease are carried off with great rapidity by septicæmia, their disease having possibly escaped recognition.

Dr. Bristowe, in 1862, laid an important communication upon the subject of acute septic ostitis before the Pathological Society of London, in which he alluded to the " acuteness, the danger, and the obscurity of the disease," and he remarked that the affection is not only deadly but common. He was not referring to the acute inflammation as attacking the upper end of the femoral diaphysis. Had he been doing so he would not have spoken of it as being " common," though he might well have described it as " deadly." Of the seven cases to which he referred, " five died speedily of pyæmia," three of them having been mistaken for rheumatism.

If only the upper end of the femoral diaphysis were attacked by the disease, how easy it would be to mistake the arthritis for a rheumatic affection, and especially so if it were associated with pyæmic purulent pericarditis !

Prognosis.—If the case is left without active treatment, abscess may extend towards the skin and effect its escape

either with or without tardy help. Or suppuration may extend along the femur or about the pelvis, and the patient may sink from blood-poisoning and exhaustion. Pyæmic abscesses may form about other diaphyses or in other joints, or pneumonia may end the distress. Indeed, in every case the outlook is exceedingly grave, for the disease is infective, and intensely pyæmic.

Treatment.—The treatment indicated in these cases is as clear, and the need of its adoption as urgent, as when the septic inflammation attacks, say, the upper end of the tibial diaphysis. In the latter case the surgeon does not wait for fluctuation; he cuts down upon the tender tissue, and whether he finds pus or not, he makes a liberal opening into the bone and uses freely his gouge, his scoop, and his germicides. He knows that want of energy or lack of thoroughness is likely to involve the entire tibial diaphysis in septic thrombosis and suppuration, and in necrosis,—to say nothing of more remote contingencies. I do not mean that when the upper end of the femoral diaphysis is implicated the line of treatment should be exactly the same. The circumstances widely differ, in that in the former case the hip-joint itself is implicated, whilst in the case of the tibia the knee-joint is unlikely to be involved, at any rate for a time, because the septic diaphysial tissue is not in communication with the joint.

If the upper end of the femoral diaphysis could be effectually dealt with without opening the hip-joint, cutting, gouging, and scraping might perchance suffice, (as in the case of the tibia), but the hip-joint being of necessity opened, and already involved in septic inflammation, it is better to remove the chief part of the intracapsular portion of the femoral diaphysis with a keyhole saw, to thoroughly disinfect the articular area with a hot solution of zinc chloride (10 grs. to the ounce), and, leaving in a large drain, to close the surface-wound only so far as seems expedient. Thus the treatment of hip arthritis which is started by septic femoral ostitis differs

widely from that of tuberculous disease of the joint, for the latter case not being septic, the surgeon closes the wound when he has completed his resection or arthrectomy. After operating for septic arthritis, however, he is compelled to make provision for drainage, for he cannot think that he has got rid of or disabled all the teeming micrococci.

If the septic focus is entirely removed with the resected portion of the femur, or rendered harmless by the erasion and irrigation, the patient may not only be completely and promptly restored to comfort, but may make an immediate recovery, as in the case which I will first record.

CASE 1.—Last spring a female infant of twelve months was admitted into the Children's Hospital, Great Ormond Street, with acute inflammation of the left hip. She was the youngest of five children, and Mr. Templeton's notes say that there was no history of tuberculosis or syphilis in the family. Till six weeks previously the infant had had no illness, but she was then treated for bronchitis. " Slight improvement took place, till the left thigh became quite useless and the child screamed when it was moved. Swelling and redness gradually appeared, and the limb became flexed." The mother also said that at this time the child became exceedingly restless, that at night she was convulsed, and that she was constantly screaming.

On admission.—The baby was ill-nourished, and lay in a rigid, semi-conscious condition. The left thigh was swollen, abducted, flexed, and fixed. Lordosis was present, and there was some deep fluctuation near the hip. At my request, the house surgeon, Mr. Miskin, made an incision over the front and outer side of the hip, evacuating an offensive abscess, the pus of which was afterwards found to be swarming with staphylococci. On exploring, the finger went straight into the hip-joint and found the head of the femur detached from the diaphysis. This having been removed, the joint was cleaned, and irrigated with mercuric solution ; a gauze drain was introduced, and

the limb was fixed on a splint. No complication ensued, and the child was sent home well and strong six weeks later, with the prospect of movement at the joint.

All cases of acute septic osteo-myelitis of the upper end of the femoral diaphysis do not, unfortunately, turn out as well as that just recorded. Here is the report of a very disastrous one, for instance, which recently came under my care at St. Mary's Hospital. It is a typical case of fulminating hip disease.

CASE 2.—On Sunday, May 12th, 1895, a schoolboy of 8½ years was admitted into St. Mary's Hospital in an unconscious and delirious condition. He kept on talking in an excited way to imaginary persons. He lay flat on his back with his eyes half closed, and his hand resting on his left groin, where the skin, though white, was somewhat œdematous. There was obscure subjacent fulness, but no fluctuation. He could not bear any pressure near the hip, and he screamed when the thigh was moved. His temperature was 104·5°, and his pulse was running, at 160. His mother said that on May 9th (three days previously) he had complained of pain and stiffness in the left hip, for the relief of which she had in vain resorted to poulticing. Delirium had come on that (Sunday) morning, and whilst raving the boy had said that he had some time previously received a kick over the outside of the hip. The mother, however, knew nothing of any such injury, and she had not seen any mark or bruise about his hip. Immediately on the boy's admission that Sunday afternoon, the house surgeon, Mr. George Riddick, asked me to come round to see an urgent case of what he took to be septic diaphysitis at the hip. Confirming his diagnosis, I had the boy at once placed under an anæsthetic, and, cutting down upon the hip-joint, evacuated an acute abscess which contained about two ounces of pus. But as the movements of the joint were all smooth and free, and as I could not satisfy myself that the pus had come from the interior of the capsule, I

did not cut into the joint.   I had not the courage of my opinion ; but thinking that if, after all, the abscess were extra-articular, by further interference I might possibly inoculate a healthy joint, I contented myself with washing out the depths of the wound with a hot germi-cidal lotion, closing the incision with sutures.

Next day the boy was a little better, but he was still so bad that it was evident that I had not effectually dealt with the source of his septic intoxication ; so I opened up the capsule and then explored the neck of the femur ; I found it denuded of periosteum in the greater part of its extent, and evidently in a condition of potential necrosis. The cartilages covering the head of the femur and the acetabulum were quite healthy.   Having resected the affected part, I finished the operation by irrigating, draining, and dressing the wound.   The boy sank six hours subsequently.   As I looked back on the case I felt that I need not charge the infirmity of purpose which I displayed on the previous day with the boy's death, for he was even then so deeply under the influence of the toxic material that the outlook was well nigh hopeless. I cannot, however, refer to the case without self-reproach.

CASE 3.—The third illustrative case is an example of acute septic hip disease started by osteo-myelitis of the femoral diaphysis, which was itself a secondary pyæmic manifestation.   As is well known, when septic osteo-myelitis attacks the end of a diaphysis, other diaphyses are apt to be similarly involved.   So common, indeed, is this scattering of the pyæmic micro-organisms in children and young persons, that some authorities speak of the disease as " multiple necrosis," which is, however, only the *result* of the disease.   Necrosis is not itself a disease ; septic Haversian thrombosis is the disease, and necrosis is the *result* only.

A boy of about ten years was admitted to the Children's Hospital, October 16th, 1894, with a high temperature

and with severe pains along the right tibia, the result of
acute septic inflammation of the growing tissue at the
upper end of his tibial diaphysis. He told us that some
weeks previously he had received a severe kick over the
upper part of the shin. This kick had, doubtless, lowered
the vitality of the growing tissues at the end of his
tibial diaphysis, and, as a result, the streptococci circulat-
ing in his blood-stream had seized upon and infected that
part. We cut down upon the end of the diaphysis, and
in cleaning out a septic abscess cavity there we removed
a small piece of dead bone. Everything went on satis-
factorily, and the wound granulated steadily, till one
day (Tuesday) he complained of pain in his left hip,
and his temperature began to rise. Knowing that the
micrococci had already undergone a successful cultivation
in his tibial diaphysis, we suspected that the same thing
was obtaining in the end of his femoral diaphysis, nor
had we long to wait before our fears were realised. I
examined him on the Wednesday, and again on Thursday
and on Friday, but could discover nothing definite or
convincing. But on the Saturday I received an urgent
message to the effect that the boy was extremely ill, and
that his symptoms now clearly pointed to the presence of
an acute septic inflammation at the end of his femoral
diaphysis. We therefore put him under an anæsthetic,
and, cutting down into his hip-joint, found around the
upper end of the femoral diaphysis a small acute abscess
which was in connection with an ulcerated patch of bone
just below the junction-cartilage. Probably the pus was
tightly locked up underneath the cervical periosteum,
which is there thickened by reflected fibres of the capsular
ligament, and by the synovial membrane passing up
towards the head of the bone. We evacuated the abscess
and excised the upper end of his femoral diaphysis. His
general condition at once improved immensely, but he
subsequently had some recurrence of disease at the part,
and his wound was long in healing. But in due course it
completely healed.

CASE 4.—Some time ago a girl about 16 years old was admitted into one of the medical wards of St. Mary's Hospital with what was thought by the house physician on duty to be acute rheumatic synovitis of the right hip-joint. She was extremely ill; she had great tenderness about the joint, and she could not bear the limb to be touched. Her temperature was high. Though she was treated with full doses of salicylic acid her condition got worse rather than better ; she was sleepless and delirious. The visiting physician asked me to see her with him, and we agreed that it was a case demanding active surgical treatment. She was, therefore, taken into the theatre, and on cutting down into the joint I found an acute abscess. There was no caries of the surface of the head of the femur; I did not excise, but contented myself with irrigating the joint and draining. Had I then better understood the pathology of these cases I should have removed the head of the femur, together with the upper end of the diaphysis—that is, most of the neck of the bone within the capsule. But, as I say, I left it, and had ultimately to resect it. Eventually the girl made a complete recovery, her convalescence being interrupted, however, by attacks of inflammation and suppuration recurring at the hip.

I am quite prepared to hear it stated by any one who may do me the honour of discussing this paper, that he is fully alive to the fact that there is a distinct and often fulminating variety of disease of the hip-joint which is due to septic inflammation of the neck of the femur, and that every surgical Fellow of this learned Society ought also to know it. If so, do we all fully recognise the lethal nature of this form of disease, and are we fully prepared to urge the immediate adoption of the serious operation by which alone the subject of it can be freed from the septic focus ?

But if every surgeon does understand and appreciate the pathology and treatment of the disease, I will shift my ground, and will say that this paper was not written for

surgeons only, but was intended to attract the attention of physicians, who are not infrequently called upon to diagnose and prescribe in these cases. Patients are not always correctly " sorted " before they come under the care of physicians, so that, when a physician sees one of these cases for the first time, and is told that his advice is being asked, for instance, for a girl of about eighteen years, who comes of a rheumatic stock, who has an acute inflammation in a hip-joint, a high temperature and pericarditis, he might by chance make an incorrect diagnosis. I do not, of course, say that he would—*might* is the word. Possibly I should not have ventured to use even this auxiliary verb, if our late friend Dr. Bristowe had not said that, in addition to "rheumatism," he had known the symptoms of the disease in question taken for those of typhoid fever and delirium tremens. At any rate I think that the importance of the subject is a sufficient excuse for my venturing to bring it before the Society.

(For report of the discussion on this paper, see ' Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. xi, p. 19.)

ON AN IMPROVED METHOD OF TREATMENT

OF

SEPARATION OF THE LOWER EPIPHYSIS OF THE FEMUR

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It would have been impossible to prove the contention of the following paper without the aid of skiagraphy. The accompanying illustrations, although apparently diagrammatic, strictly represent (with the exception of Fig. 1) the skiagraphs made for this purpose. The outlines of the bones, &c., were accurately traced from the latter, and reproduced on a smaller scale. We are much indebted to Mr. Waren Tay, Mr. Mansell Moullin, and Mr. Openshaw for permission to use cases under their care.

In practically all cases when the lower femoral epiphysis is torn off by over-extension of the knee-joint, it is carried forward on to the front of the diaphysis by the

pull of the crucial ligaments. It is then somewhat drawn up by the quadriceps extensor muscle. The thick periosteum is stripped up from the front of the femur for two or three inches, and remains attached to the epiphysis, giving it a forward tilt, so that its articular surface looks downwards and forwards. It will also be seen that the posterior edge of the lower end of the diaphysis is sharp and angular, and projects into the popliteal space perilously near the skin, and pressing on the vessels and nerves.

Our method of reduction was devised to make the epiphysis retrace its steps backwards, to apply the separated periosteum to the anterior surface of the femur, to withdraw the end of the diaphysis from the popliteal space, and when these objects have been attained to so fix the limb that the displacement cannot recur. The method is as follows :

Under complete anæsthesia an assistant makes steady but strong traction upon the tibia in the line of the limb. This overcomes the upward pull of the quadriceps extensor, and brings the epiphysis down to the line of separation. The operator then clasps his hands beneath the thigh and draws it steadily upwards, *gradually flexing completely the knee* and hip-joint.

It will be seen that this manœuvre causes the epiphysis to move back upon the fractured surface of the diaphysis until it has reached its normal position, and further movement is prevented by the periosteum coming into tight contact with the anterior surface of the femur (see Fig. 1).

A domette bandage is then applied around the thigh and ankle, fixing the heel upon the buttock, and the limb is laid upon its outer side on a pillow. An ice-bag can conveniently rest upon the front of the knee to limit effusion.

The advantages of maintaining this position for a fortnight are these :—The quadriceps exerts tension in the length of the bone, keeping the surfaces in close apposition and squeezing out effused blood. The tendon

of the quadriceps and the patella fit into the groove
between the two condyles, and prevent lateral displace-

Fig. 1.
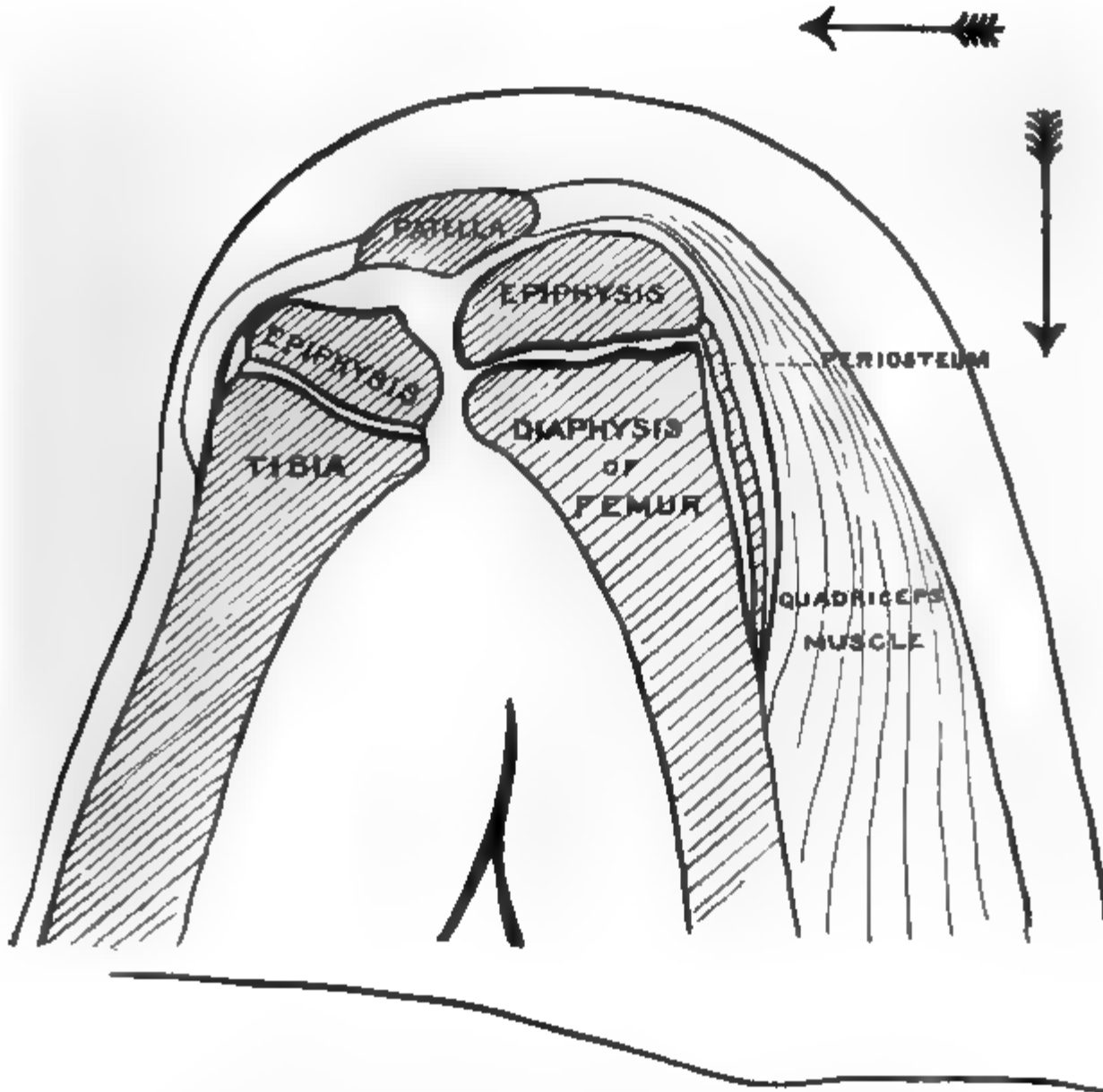
Diagram representing the method of reduction described above.   Full
flexion of the knee carries the detached epiphysis back into place in
the direction of the horizontal arrow, whilst the tension of the quad-
riceps thus produced presses the epiphysis against the end of the
diaphysis, as indicated by the vertical arrow.

ment; and since the tendon of this muscle changes its
direction at a right angle, to be inserted into the tubercle
of the tibia, it prevents recurrence of the forward displace-
ment of the epiphysis.   This position also removes the
sharp end of the diaphysis as far as possible from the

popliteal space, and relaxes the skin and vessels and nerves completely. The articular surfaces of the epiphysis are subcutaneous and uncovered, so that their position can at any moment be observed, and the anterior and posterior tibial arteries are not concealed by any splint or apparatus.

After fourteen days the limb is placed upon a Macintyre splint flexed to as sharp an angle as possible, and this is gradually opened, until at the end of another fortnight the knee is fully extended. It is then put up in plaster, and the patient is able to get about.

The plaster is kept on from a fortnight to a month; a little massage restores the movements of the joint.

It is certain, but not yet generally admitted, that weight extension is quite useless in the treatment of separation of this epiphysis. First, in those cases where the periosteal sheath is intact and the epiphysis merely loosened, there is nothing to cause displacement and to be counteracted. Secondly, where the epiphysis is much displaced, as shown in some of the illustrations, no amount of weight extension is likely to bring it into place. Thirdly, when the epiphysis has been really got back into position, weight extension is quite unnecessary in order to keep it there, since rest on almost any form of splint will be sufficient.

Once this axiom of the inutility of weight extension be admitted, we think all objections to the flexed position disappear. Relaxation of the gastrocnemius muscle, the powerful effect of direct pressure upon the epiphysis through the quadriceps muscle and patella, and thirdly, the backward traction exerted through the tibia and crucial ligaments, can only be obtained during flexion of the knee.

Theoretical considerations, post-mortem experiments, and above all the experience of actual cases, are alike in favour of the treatment by flexion. We are confident that if this method of treatment comes into general use, with the aid of the accurate control of skiagraphy, com-

plete separation of the lower femoral epiphysis will lose its dangers to the patient, and cease to be a discredit to surgery.

This applies not only to simple, but to compound cases. In the series of cases collected by one of us [1] examples are to be found where violent efforts at reduction of the displaced epiphysis were prolonged for one or more hours, and of cases in which weight extension, &c., were followed by extremely bad results. It is perfectly true, on the other hand, that nature moulds the displaced fragments into a very useful bone, and this is illustrated by at least one case now recorded ; but there is not only the risk to the popliteal vessels by compression over the diaphysial end, but the greater chance of arrest of growth in this extremely important epiphysial disc if the displacement be not overcome. Further, we are bound to aim at the best possible immediate result, and in these days, when skiagraphy is so common, the public will insist upon obtaining it.

As already noted, in nearly all cases the displacement that has to be corrected is that of the epiphysis forward with or without twisting on a vertical axis ; in other words, the diaphysis projects backwards into the popliteal space. This is exactly what we should expect from the nature of the accident, which in a large proportion of cases involves violent hyper-extension with a varying degree of torsion of the leg. If this displacement is left uncorrected, the ultimate result on the joint will be to allow of extension beyond the normal, so that the tibia and femur form an angle with each other, open in front, instead of a vertical line. More than one case could be here adduced to prove this statement, but since the hyper-extension constitutes only a slight defect, it is unnecessary to give more than one example ; this case is of interest, since the child was examined and the skiagraph obtained four and a half years after the accident (Fig. 2).

[1] Jacksonian Prize Essay, R.C.S.

FIG. 2.
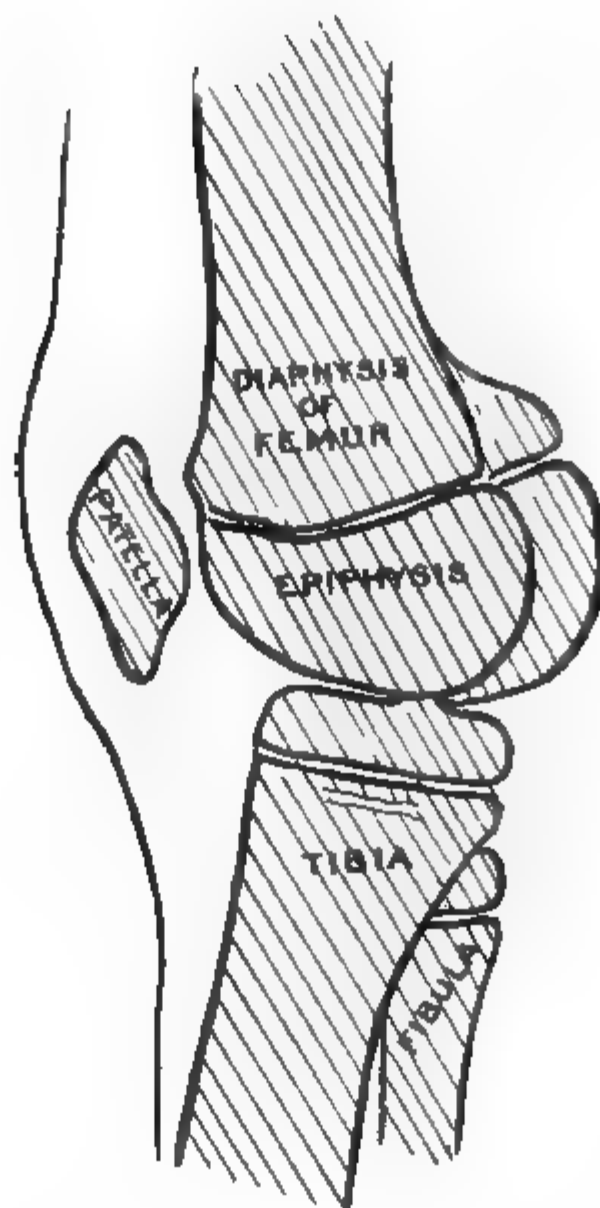
Skiagraph from Case 1, obtained four years after the accident.

CASE 1.—Sophia S—, a rickety child aged 6, fell in the street and sustained a separation of the lower epiphysis of the left femur on April 3rd, 1893. The limb was put up extended in a box splint for a month. In November, 1897, she was seen again; there was no shortening, and she could walk and run well; nevertheless the epiphysis had united in a somewhat rotated position, so that the outer condyle faced more inwards than it should, the inner condyle more posteriorly. The left knee could be hyper-extended no less than 25° more than the right one, but, as already said, the limb was perfectly useful.

We believe that the method of treatment advocated in this paper is to some extent a new one, and we are not

aware of any records of cases so treated (that is by the full flexion of the knee). This idea is due to Mr. Bar nard ; at the same time it has been held by some fo₁ long that flexion to at least a right angle is essential i₁ the treatment of cases where the displacement has beeɪ complete. Thus, for instance, Mr. Hutchinson, sen. wrote in 1888 (re displacement of this epiphysis) " When once it has occurred, such a displacement is pro bably impossible of rectification excepting by putting the limb with the knee at right angles, and in that positioɪ it is exceedingly difficult to secure the needful extension I do not know of any case in which, after such displace ment, reduction was proved to have been effected." [1]

With regard to the latter point, we maintain tha extension by weight is wholly unnecessary in these cases and further, that, as the examples now brought forwarᵢ show, there is no difficulty in getting perfect reposition o the displaced epiphysis.

In an abstract of three lectures on " Injury to th Epiphyses " one of us wrote,[2] " Before attempting t reduce the displacement it is absolutely essential tha muscular spasm should be prevented by thorough anæ thesia ; the knee should be flexed, and then traction an direct pressure brought to bear on the epiphysis. . . . When reduction has been effected the best splint to use i a carefully padded Macintyre's one, flexed to an angle ⟨ . 135°."

In a single case with typical backward projection ⟨ the diaphysis recorded by Mr. A. F. McGill (see Joh Poland's work on the Epiphyses, p. 764), " under eth₍ the deformity was easily reduced by forcible flexion of tɪ limb till the heel touched the buttock." This is the onɪ allusion to the method advocated by us that we have bee able to find.

The epiphysis, if separated, nearly always travels fo wards, whilst the diaphysis projects into the poplite

[1] ' Clinical Illustrations of Surgery,' vol. ii, p. 3.
[2] ' British Medical Journal,' 1894.

space, because the injury is caused by hyper-extension of the knee (very commonly by the leg getting caught in a wheel, and the patient being swung round by it).

In experiments on the dead subject one of us [1] found extreme hyper-extension with some twisting force to be the easiest method of producing detachment of the epiphysis. In the living subject we find some lateral displacement often complicating the forward one, and there may be rotation of the epiphysis on a vertical axis, proving the occurrence of a twisting force, but the forward displacement is the chief and most usual one; indeed, it may be said the epiphysis never travels backwards. The stretching of the popliteal vessels, and even the internal popliteal nerve, over the edge of the diaphysis, which projects backwards, has been well illustrated in Mr. Hutchinson's 'Clinical Illustrations of Surgery,' the drawing being taken from a case in which gangrene was produced.

It is of interest to compare separation (with displacement) of the lower epiphysis of the humerus with that of the femur.

We find precisely the same statement applies to both,— that the diaphysis tends to project towards the flexor aspect of the joint, where run the main vessels.

Here, again, some lateral displacement or twisting of the epiphysis may be present, but the antero-posterior one is the chief. Owing, however, to the bone being separated from the artery by the brachialis anticus, the diaphysis is less likely to press on the artery. It is easy to overlook this projection of the diaphysis forwards, and sometimes difficult to overcome it, even if recognised. Some surgeons, especially Professor Christopher Heath, have advocated putting up the arm with the elbow fully flexed, and from analogy with the knee-joint it would appear to be an excellent method.

It is not contended that all cases of separation of this epiphysis are likely to give much trouble to the surgeon,

[1] J. Hutchinson, jun.

or require any special method of adjustment. Everythin;
depends on whether the diaphysis has been forced throug:
the periosteal sheath, which it will be remembered i
continuous with the epiphysis, and in cases of separatio:
is invariably dragged off with it. It stands to reaso
that if the periosteum is merely loosened from the bone
and the epiphysis not shifted from the broad end of th
diaphysis, good union will result in whatever position th
limb is put up, and the only result of the injury may b
temporary deposit of new bone round the lower end c
the femur, with the chance of arrest of growth at a late
stage. There is no doubt whatever that many of thes
cases of detachment of the epiphysis without displace
ment are overlooked in children and young adults ; indee(
it may be extremely difficult to detect them.

The following case is an example of the condition ju:
described, and the skiagraph taken eight months afte
the accident showed that the treatment with the limb i
an extended position left nothing to be desired (Fig. 3).

CASE 2.—F. O—, a boy aged 3½, was admitted into the hospit
on February 21st, 1897, having sustained a fracture in the midd
third of his left femur, with a separation of the lower epiphysis
the same bone, without any displacement. The limb was put ι
in a box splint with weight extension. At the end of five weeks]
was sent out in a poroplastic splint. On November 1st, 1897, tl
boy was brought up for inspection and the skiagraph obtaine
He could then walk and run well. There was neither shortening
the limb nor over-extension of the knee.

Before adducing other original cases we will quo·
illustrations of the difficulties that have been met with :
the treatment of this injury where the displacement h:
been complete.

M. Ardouin lately reported[1] an unsuccessful attem]
to save a limb in a case of compound separation of tl
lower femoral epiphysis. The patient was a lad ag(
sixteen, whose leg was caught in some printing machiner

[1] ' Bull. de la Soc. Anat.,' June, 1897.

and he was carried round by it. Through a large wound at the back the lower end of the diaphysis protruded.

FIG. 3.
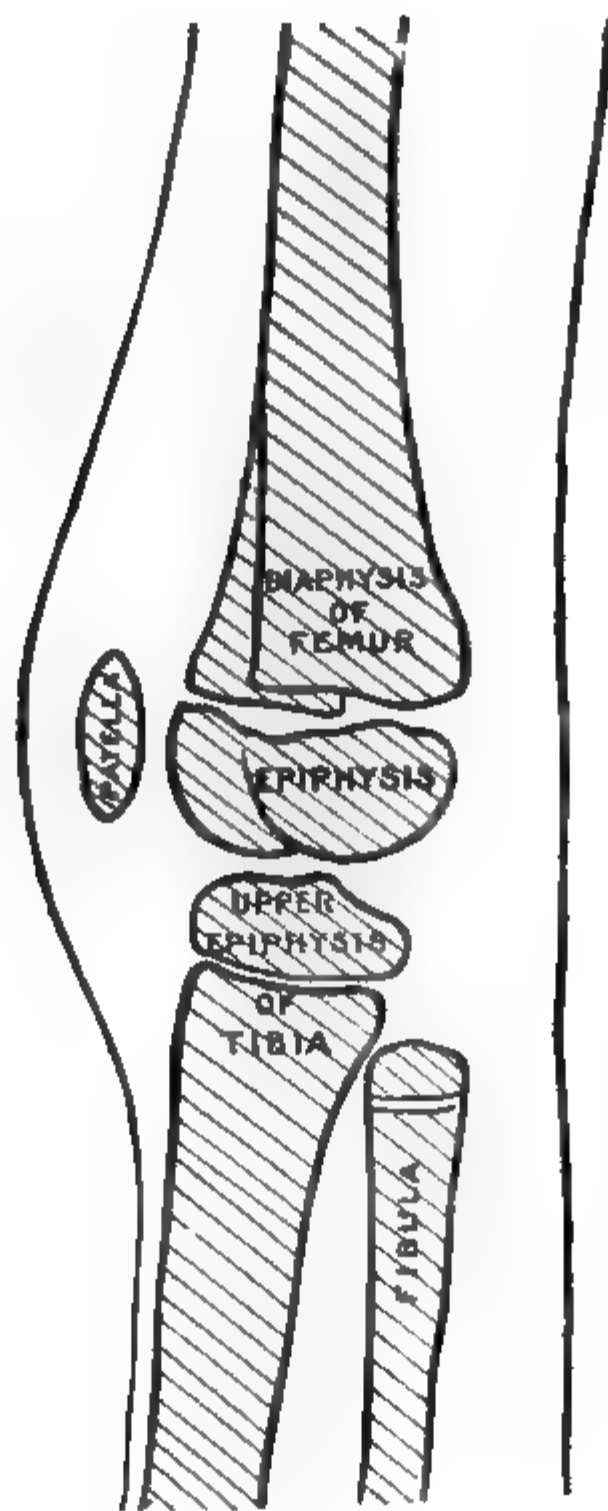
Skiagraph obtained nine months after separation of the epiphysis without displacement (Case 2).

After reduction (no details are given as to the method of treatment employed) things appeared to go on well fo r a

fortnight, when it became necessary to amputate the limb, after which operation the boy recovered. It was found that the projecting diaphysis had torn open the popliteal vein, and that much hæmorrhage had occurred. Further, there was an abscess in the wall of the popliteal artery.

Such a case, where one or both main vessels in the popliteal space are seriously damaged by the projecting diaphysis, is probably doomed to amputation from the first, and it is to be feared that this will, with rare exceptions, in spite of the advance of surgery, remain true for long. But one must discriminate, and we believe the prognosis would be less grave than has been just stated if two simple rules were observed in the treatment of these cases. Before giving them we shall be excused for dwelling on a few preliminary points.

First, as regards the gravity of compound separation of the lower femoral epiphysis the following figures[1] will convince anyone that it has not been exaggerated. Out of thirty cases collected the result was—

Death from shock 4 (13%)
Primary amputation, of which at least 3 cases died . 13 (43%)
The diaphysial end was resected and reduction thus secured—all recovered with useful but somewhat shortened limbs 5 (17%)
Reduction was more or less effected without resection; of these 3 died from pyæmia, 4 required subsequent amputation, and one only recovered, retaining his limb after prolonged suppuration and necrosis . . . 8 (27%)

Thus, out of the total of thirty cases, ten at least proved fatal (*i. e.* no less than 33 per cent.), whilst of the remainder fourteen recovered only at the cost of the limb (in one case amputated at the hip-joint), and only six (20 per cent.) recovered without this mutilation.

Is there any other compound fracture of similar frequency in which such depressing results follow the sur-

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[1] J. Hutchinson, jun., ' Injuries to the Epiphyses,' Jacksonian Prize Essay, R.C.S. See also ' Brit. Med. Journ.,' 1894.

geon's best efforts at treatment ?   And it must be remembered that all the patients are young and probably healthy lads.   It is no exaggeration to say that a compound fracture of the skull, if properly treated, is in itself of little importance compared with a compound separation of the lower epiphysis of the femur.

Of course, much better results are obtained when no wound has complicated the separation of the epiphysis. But even here there is very much to be desired.   Thus, out of thirty-four cases which one of us observed or collected records of,—

Good reposition was obtained, and recovery with very useful
  or perfect limbs resulted in 19 (57%)
Imperfect (we may say very imperfect) reduction in   . 15 (43%)

Of these sloughing or suppuration followed in no less than . 6
And every one of these required an operation:
  Amputation through the thigh in 4
  Excision of the knee-joint 1
  Resection of the end of the diaphysis   .   .   . 1

It should also be noted that in more than one of the cases in which suppuration did not follow, the backward displacement of the diaphysis produced such trouble from the pressure on the popliteal vessels, &c., that an operation had to be done (chiselling or sawing off the end). In one famous case (Sir C. Bell's) the projection of the diaphysis led to an aneurism of the popliteal artery by its pressure on the wall of the vessel. This occurred twenty years after the original separation of the epiphysis, and amputation then became necessary.

Those who have not encountered cases where the epiphysis has been completely displaced, and the diaphysis projected through the periosteal sheath, will hardly credit the extreme difficulty that may be met with under such circumstances in procuring reduction by traction and direct pressure in the extended position of the knee.

Many cases could be quoted in which repeated or prolonged attempts under anæsthesia have quite failed (in some of these amputation has been resorted to), and

many others in which reduction has been supposed to have been effected, but the displacement was said to have recurred subsequently. In the latter case it is safe to say that a proper reduction had never been made. We believe that such difficulty in reduction, even in compound cases, will be almost, if not quite, removed by the adoption of the full flexion treatment.

A few typical cases will here suffice.

In the well-known case recorded by M. Delens,[1] a boy aged eight had a compound separation of the epiphysis, the diaphysis projecting through the skin on the outer side of the popliteal space ; prolonged efforts at reduction under chloroform proved unavailing, both on the day of accident and the following one, so that it became necessary to resect the end of the diaphysis by means of a saw.

In a case under the care of Dr. McDougall, of Carlisle, a man aged twenty had simple separation of the epiphysis ; so much difficulty was experienced in effecting reduction that the attempt had to be abandoned, and as at the end of a month the skin had ulcerated over the projecting diaphysis, whilst no union had occurred, amputation was done.

In a case under the late Mr. Gay of a lad aged 13, there was protrusion of end of diaphysis through wound over popliteal space ; failure of all attempts to reduce the displacement under anæsthetic ; resection of diaphysial end necessary.

In a similar case under M. Reverdin,[2] although the projecting end was in part cut off, the reduction under full extension was evidently quite incomplete, since gangrene ultimately followed from thrombosis of the popliteal artery, due to pressure on it of the diaphysial end. M. Reverdin's account is a very full one ; he candidly admits the imperfect reduction was to blame in the loss of the limb, and whilst he gives a good general review of the subject,

[1] 'Archives gén. de Médecine,' 1884, p. 272.
[2] 'Revue Médical de la Suisse Romande,' 1886, p. 291.

he makes no mention of any other position in the treat-ment than full extension.

In a case recorded by Hamilton,[1] a doctor's son sustained compound separation, and it was only after an hour's vigorous efforts on the father's part that some sort of reduction was effected. After some necrosis the boy recovered with a shortened leg and an ankylosed knee-joint.

It is unnecessary to extend the list further. Examples could be adduced where gangrene of the limb, and even death of the patient, has followed even in simple cases of this displacement, which have been either diagnosed wrongly, or forcibly and unavailingly manipulated in the extended position. Thus in the case of a boy aged thirteen, treated by M. Richet,[2] the diaphysis projected through a wound; reduction was effected (or supposed to be effected) by violent and prolonged traction and extension, with the aid of a spatula introduced through the wound. This instrument broke, and was replaced by forceps used as a lever. The displacement had recurred before the child's death on the eighth day from pyæmia.

A consideration of these facts must favourably incline surgeons to adopt any method which promises better results. From the cases we now bring forward we believe it will be found that all such violent manipulation is unnecessary, and that the resection of the diaphysial end will very rarely be required. Of course everything depends on correct diagnosis being made early, and unfortunately, owing partly to the belief that separation of the epiphyses is too rare an accident to be worthy of careful study, and to the wholly inadequate manner in which the subject is treated in most of the text-books, mistakes in the diagnosis are very apt to occur.

The skiagram from which the following figure was made was taken two months after complete separation of the lower epiphysis, and is of particular interest since the

[1] 'Fractures and Dislocations,' p. 530.
[2] 'L'Union Médicale,' 1876, p. 426.

case had been under treatment in hospital during the whole of this time, and the extensive displacement had not been made out before the photograph was taken.

CASE 3.—William R—, a schoolboy aged 8, whilst riding on the back of a carriage got his right leg entangled in the wheel, and was carried round by it. He was brought to the hospital the same day,

FIG. 4.
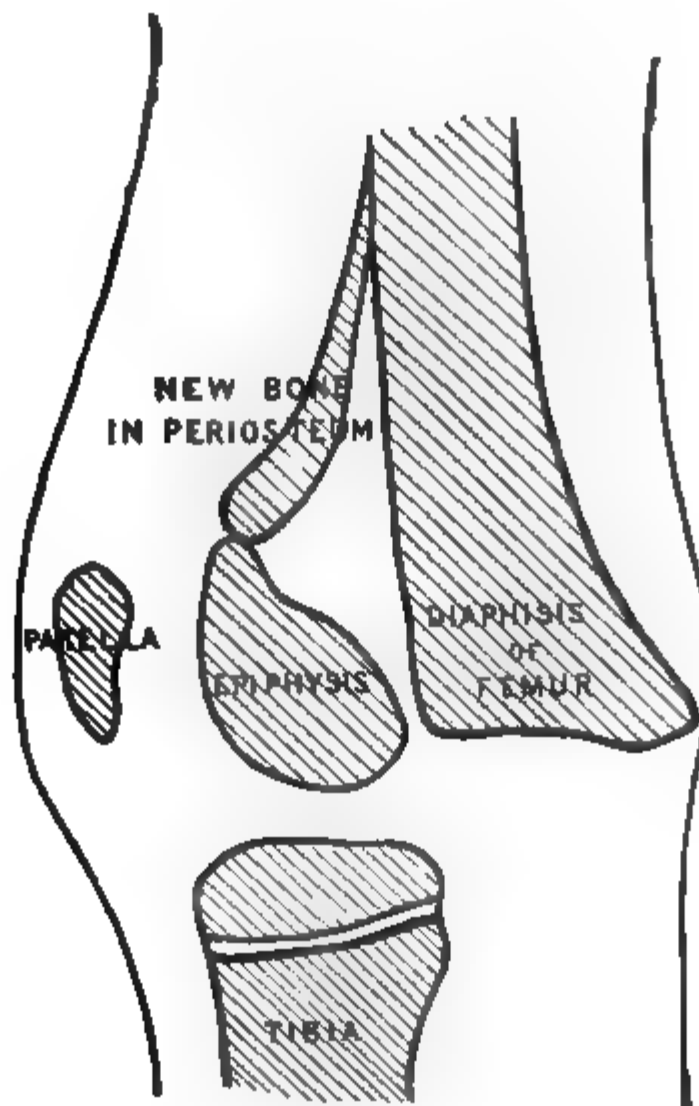
Skiagraph from Case 3, taken after two months' treatment with weight extension, showing backward displacement of the diaphysis with new bone connecting it with the epiphysis.

and on examination the lower epiphysis of the femur could be felt to project forwards, the diaphysis backwards into the popliteal space; abnormal lateral mobility and some creaking were present. The limb was put up in a box splint with weight extension; at the end of eight weeks, when the patient was up on crutches, a skia-

graph was made.   This showed that the diaphysis only touched the
epiphysis at the posterior edge of the latter.   The epiphysis itself
was tilted forwards, and running up from its upper border to the
anterior surface of the femur was well seen the periosteum, which
had already ossified, and was practically the only means of union
between the two (see Fig. 4).   It is remarkable that with so great a
backward displacement the circulation in the popliteal vessels was
not wholly obstructed.   It was thought best to operate, especially
since the power of flexion in the knee was very limited.   There was
then only one eighth of an inch shortening.

Accordingly Mr. Openshaw operated by an incision on the inner

Fig. 5
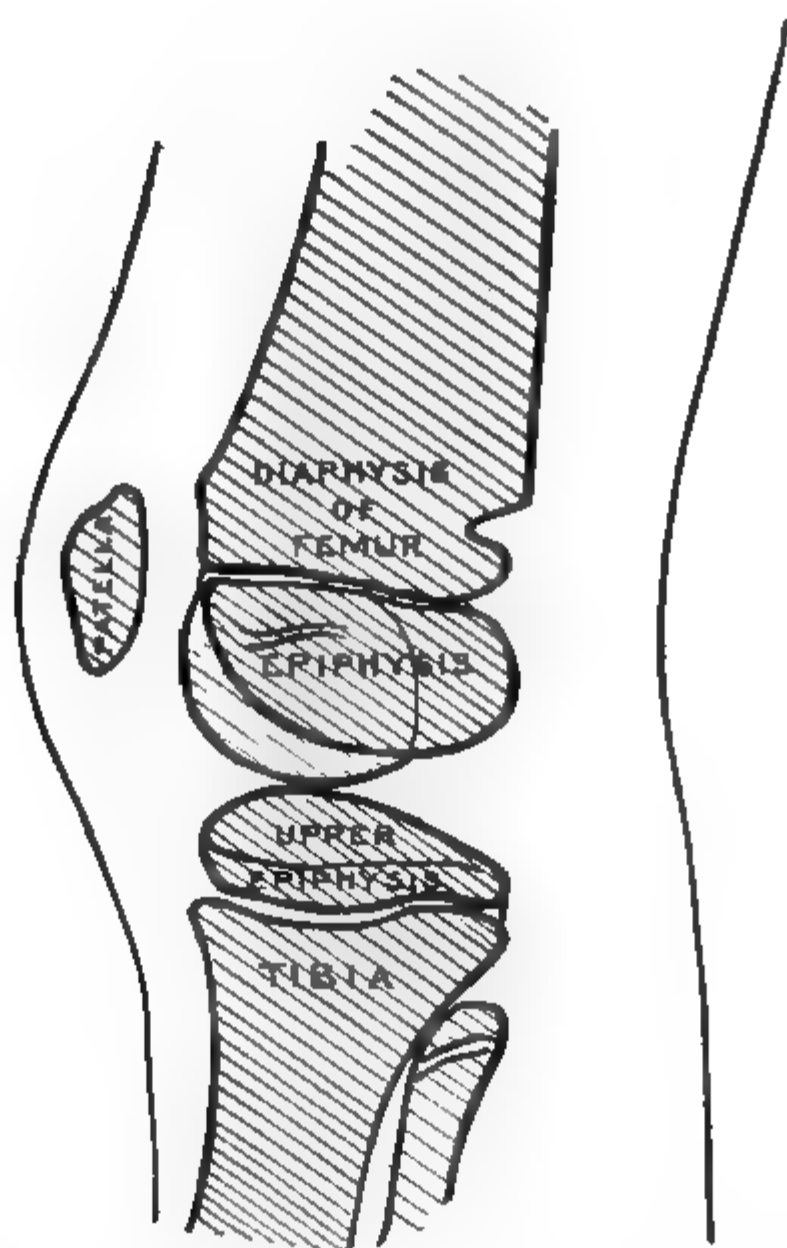
Skiagraph from Case 3, taken seventeen months after the operation,
showing that the new bone has formed an almost normal femur-end.

side of the outer condyle; one inch of the lower end of the diaphysis was chiselled off, so that the knee could be flexed to an angle of 140°. The wound healed perfectly, and the patient left the hospital much improved. This was in June, 1896. In November, 1897, seventeen months later, the patient was got up for inspection, and a second skiagraph made. This shows in what a really marvellous way the bone has been re-formed, so that hardly anything abnormal can now be noticed in the skiagraph (Fig. 5). The knee cannot be hyper-extended; he can run and walk well, but wears a thick sole on his boot to counteract the shortening of three quarters of an inch. The surface of the condyles is at right angles to the axis of the bone, but is displaced somewhat forwards, so that in full flexion of the knee the upper border of the patella only reaches over one third of the condyle. It will be seen from the above measurements that no arrest of growth has occurred, since the limb is only three quarters of an inch shorter, and at least this amount of bone was removed at the operation.

This illustrates a fact brought out strongly in a series of experiments on epiphysial detachment in lower animals made by one of us some years ago. It was then found that the epiphysial disc, consisting as it does only of columns of modified cartilage cells, is remarkably difficult to destroy, and will sometimes remain for long unaltered, although removed from its normal place at the end of the diaphysis. It would appear that this holds true perhaps even more in the case of man than in the lower animals. It may be noted that the life of the epiphysial disc in a rabbit is measured only by months instead of years in the human subject.

CASE 4.—Joseph J—, aged 9, had the right thigh run over by a van wheel, February 2nd, 1896. The accident was followed by much effusion into the knee-joint and swelling around it, and hyper-extension of the leg on the thigh could readily be obtained under an anæsthetic. There was no definite crepitus. The limb was treated in the extended position on a splint for six weeks, and after some time a positive diagnosis of separation of the lower epiphysis was arrived at. This was fully confirmed by the Röntgen rays. The accompanying reproduction of the skiagraph shows the epiphysis displaced forwards with the tibia, the diaphysis touching it only at one point and projecting strongly into the popliteal space (see Fig. 6). As the swelling went down the projection of

bone could be plainly felt. The boy was in hospital eighteen weeks. When he left he could walk fairly, but the power of flexion

FIG. 6.
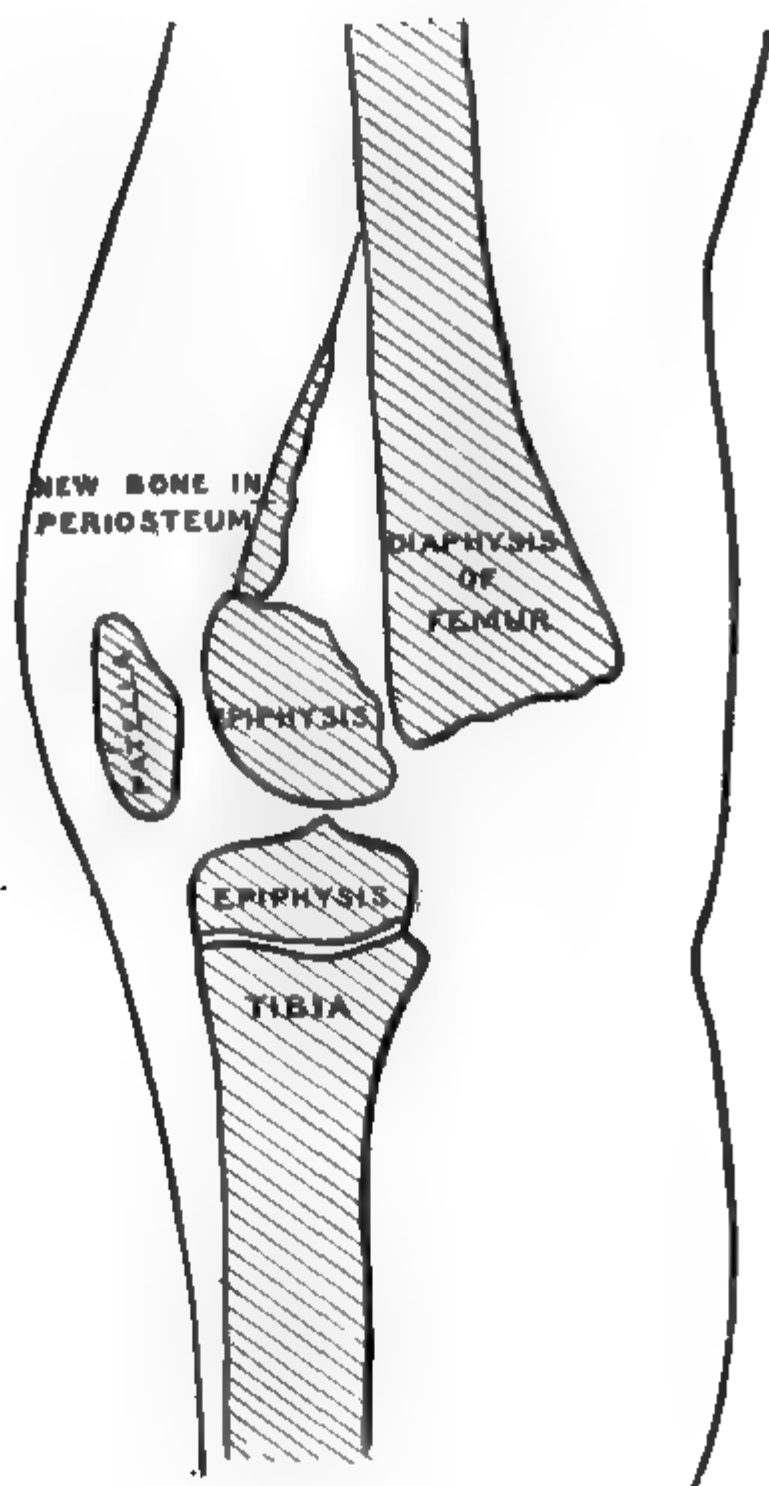
Skiagraph from Case 4, treated with weight extension, showing persistent backward displacement of the diaphysis.

was extremely limited, and some hyper-extension was present. It has been found impossible to follow up this patient.

The following case illustrated the excellent result to be obtained from the treatment by full flexion.

CASE 5.—Arthur W. M—, aged 11, was running behind a cab and attempting to jump up behind, when his right leg caught between

FIG. 7.
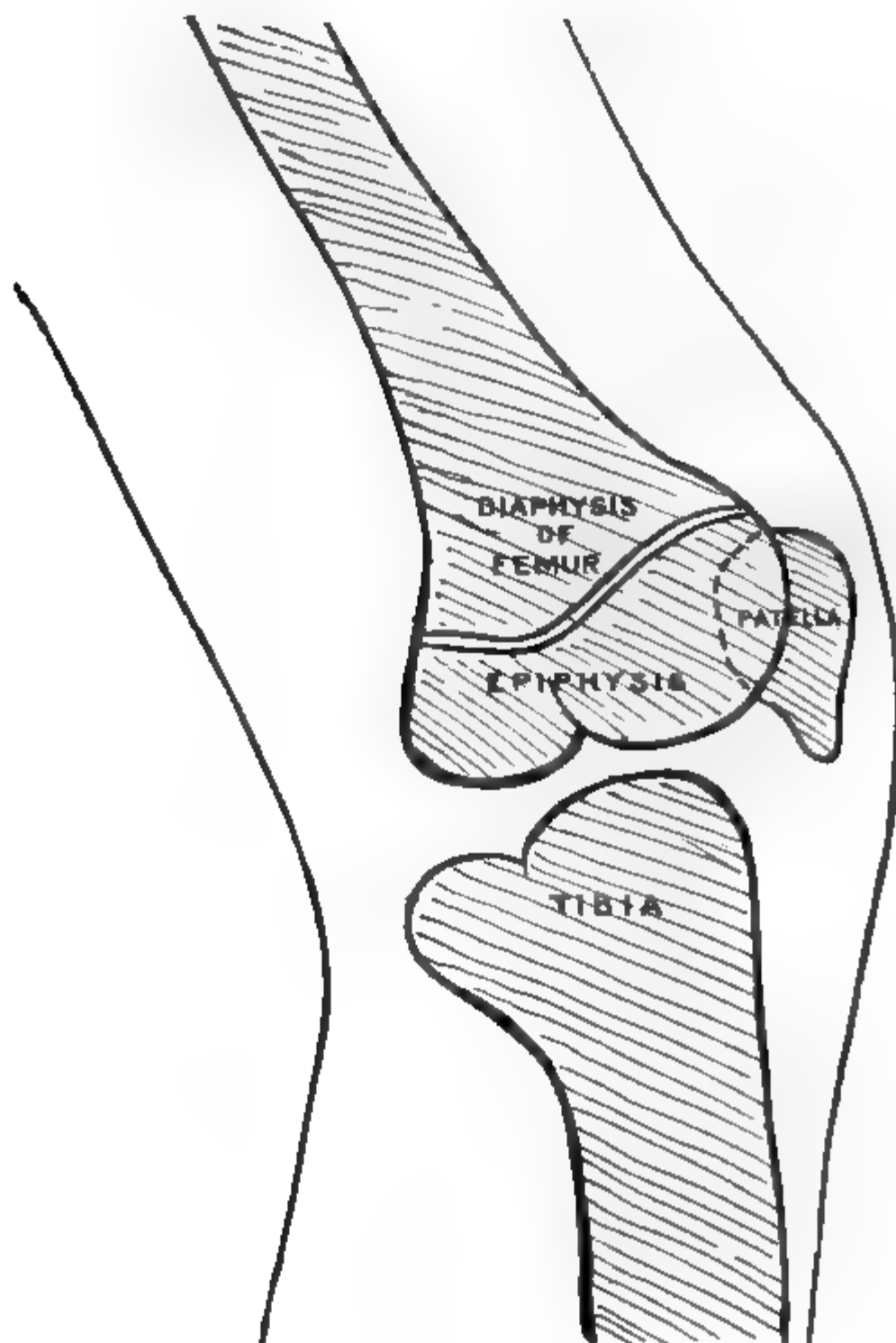
Figs. 7 and 8 show the result of treatment by full flexion in Case 5, the epiphysis being in perfect position.

the spokes of the wheel, and he was carried round by it until the
vehicle was actually stopped by his limb catching against the
spring. After the accident he was much collapsed. On admission

Fig. 8.
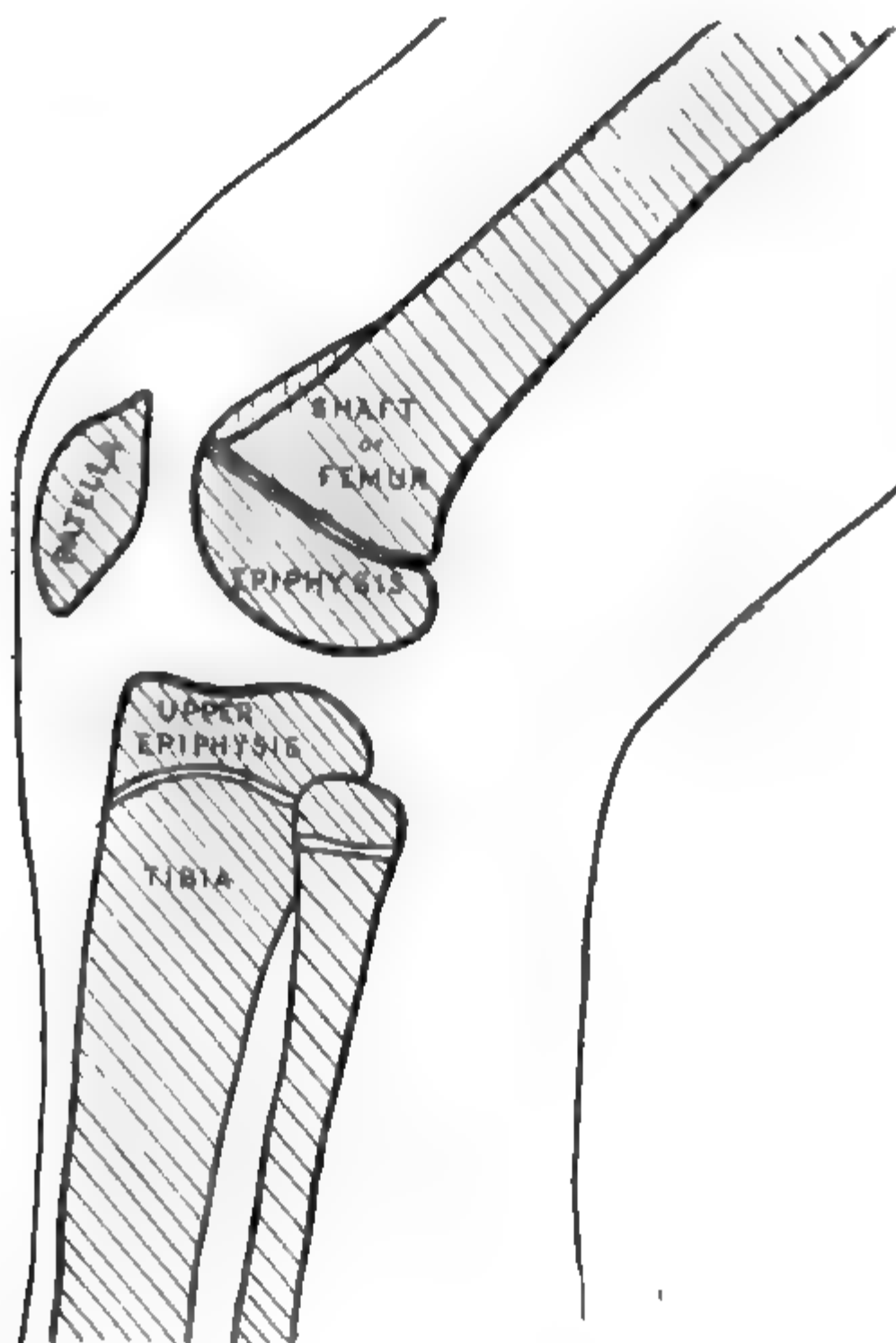
to the hospital the house surgeon, Mr. O. B. Howse, anæsthetised
him and readily made out a complete separation of the lower
epiphysis of the femur, the diaphysis projecting back into the

popliteal space. Traction was made on the leg in the extended position and kept up, while another person flexed the hip and knee by clasping his hands under the thigh and slowly elevating it. A soft leathery crepitus was felt. The limb was then fixed by a bandage in the fully flexed position, and an ice-bag applied over the flexed knee-joint.

A small dose of morphia was given, but there was no great discomfort after reduction. At the end of ten days the bandage was removed and the limb put up on a Macintyre's splint flexed to a right angle. The epiphysis remained in excellent position, and was firmly united at the end of a month from the accident, when he was sent out of the hospital. Before he left a skiagraph was taken, and although it was made somewhat obliquely (see Fig. 7), so that the bones appear a very peculiar shape, it can yet be made out that the epiphysis is well in position.

A later skiagraph, taken from the side (see Fig. 8), shows that the result could not be improved upon. It is necessary to emphasise the facts that in this case complete forward displacement had occurred, that the reduction was quite easily effected, that no inconvenience resulted from the fully flexed position maintained for ten days, and finally, that good union without damage to the epiphysial line speedily resulted. At the end of two months from the accident his condition was as follows:

The two limbs were of exactly the same length; he could run and walk well, and there was nothing abnormal about the right knee except very slight limitation of flexion and a little thickening above the epiphysial line.

The following case, which was under the care of Mr. Waren Tay, illustrates the ease with which a completely displaced epiphysis was got back into position and maintained there in full flexion. This position was kept up for sixteen days.

CASE 6.—*History.*—Fell down crossing a road, and a brougham passed over his left knee. He was brought on a stretcher.

Present state.—The femur had gone back into the popliteal space, and was just beneath the skin. The tibia with the epiphysis was dislocated forwards and rotated.

Treatment.—The patient was put under ether, and the leg pulled upon and rotated into position. The thigh was then gradually flexed upon the belly whilst extension was still kept up on the leg until the knee was fully flexed, when it was fixed in that position by a bandage applied round thigh and leg. It was then placed upon

ts outer side on a pillow. Morphia one sixth grain given. The arteries of the foot were watched: Lotio Plumbi applied to the knee.

FIG. 2.
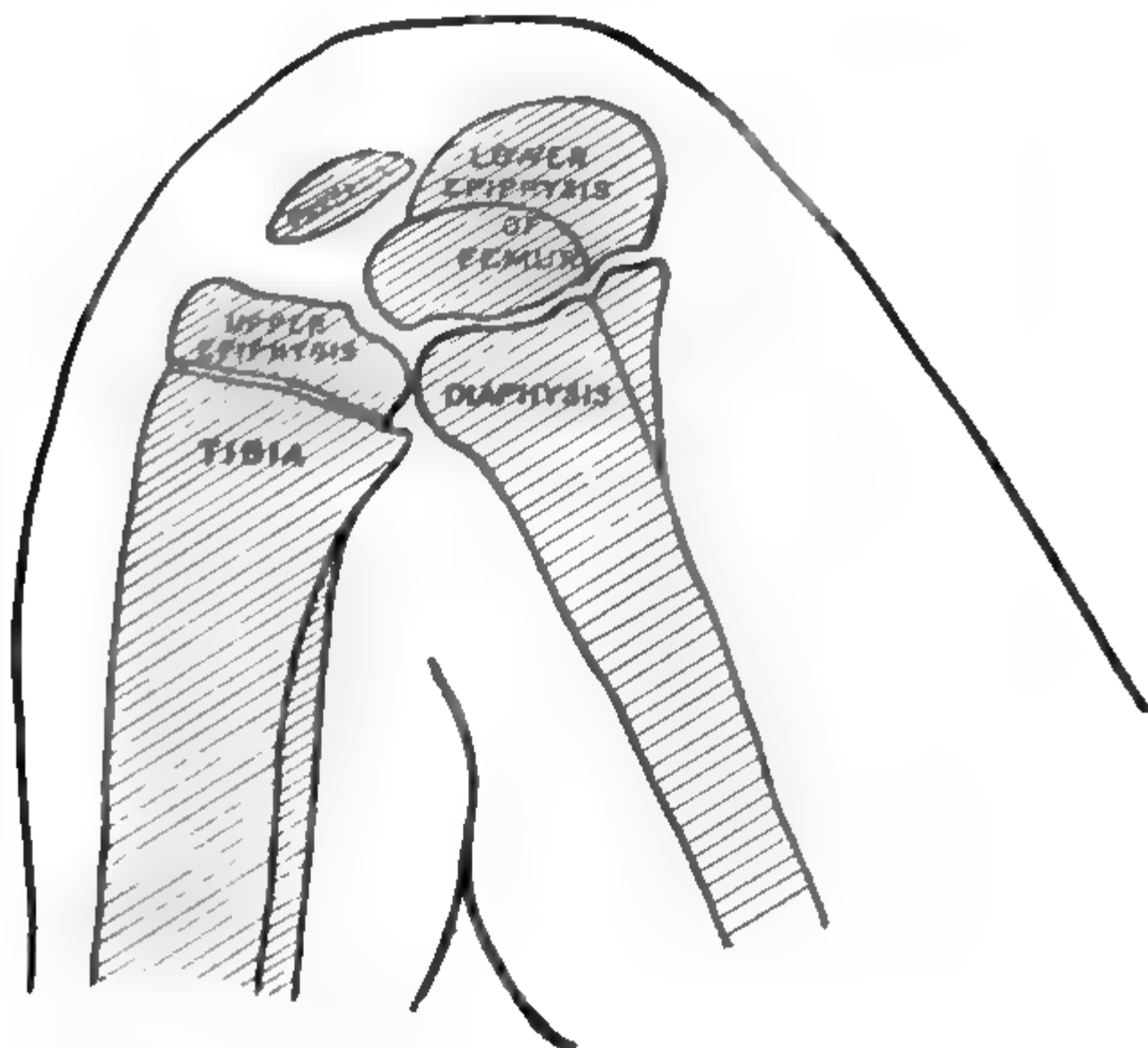
Skiagraph from Case 6, taken shortly after reduction of the displacement by full flexion of the knee.

October 1st, 1897.—Great swelling and ecchymosis, especially in popliteal space. A skiagraph was taken a day or so after the accident (see Fig. 9). The swelling gradually went down.

15th. The leg was taken down and put up on a Macintyre at a right angle. It had been totally flexed for sixteen days.

The Macintyre was then gradually opened until it was in a week or so quite straight.

It is of especial interest to follow up cases of separation of this epiphysis years after the accident has occurred,

but few records of such observations have been published. We are fortunately able to give four such cases besides the one described already (Case 1).

Case 7.—Charles G—, aged 14. December 18th to March 31st, 1888. In hospital 103 days, with compound fracture of skull and separated lower epiphysis of right femur.

History.—Fell down a lift hole 30 to 40 feet.

Examination.—Right leg, some effusion (probably blood) into knee-joint. Right anterior superior spine to internal malleolus = 25⅗ inches; left = 27⅗ inches, hence 2 inches shortening.

During the night and next morning he complained of a good deal of pain in the right leg and foot, which was very markedly colder than the left one, though pulsation could be detected in both anterior and posterior tibial arteries at the ankle.

December 19th.—Right knee 14 inches round; left knee 11¾ inches round.

Right leg.—Distinct lateral movement under anæsthetic. The lower border of projection behind is most prominent on the outer side and level with the tubercle of the tibia, and runs in a regular line for about three inches across the popliteal space.

This abrupt projection is evidently the lower end of the diaphysis, and it does not appear to move with the leg on moving that laterally, whereas the epiphysis (whose upper margin corresponds to about the upper margin of the patella) distinctly moves with the leg. No rotation or flexion of the epiphysis can be made out, though of course an exact examination is difficult owing to the swelling.

The knee being somewhat flexed, steady extension was made on the leg whilst the end of the diaphysis was pressed forwards. At first no yielding was obtained, but after a few minutes the diaphysis was felt to return into its place with a cartilaginous " rub," no bony crepitus whatever being felt. On measurement the limb was found to be 27 inches. It was then put upon a Macintyre splint flexed at an angle of 150°.

22nd.—The posterior tibial pulsation is much more vigorous in the right leg than before. There seems to be still some tendency for the diaphysis to be displaced backwards, but it is very difficult to be sure of this, owing to the effusion of blood into the knee-joint lifting up the patella. Ice-bag still applied to knee.

January 4th.—Leg taken down from splint, each measures 26½ inches, no shortening, but a little increase of circumference (½ inch). No displacement detected; the union already seems very fair.

29th.—One third inch or a quarter inch increased circumference in right knee, due to thickening round the bone; both legs precisely

the same length; can flex knee to a right angle, beyond this some pain.

The patient was examined by Mr. Barnard in Feb., 1898, and a skiagraph (Fig. 10) taken. The epiphysis of the femur has com-

FIG. 10.
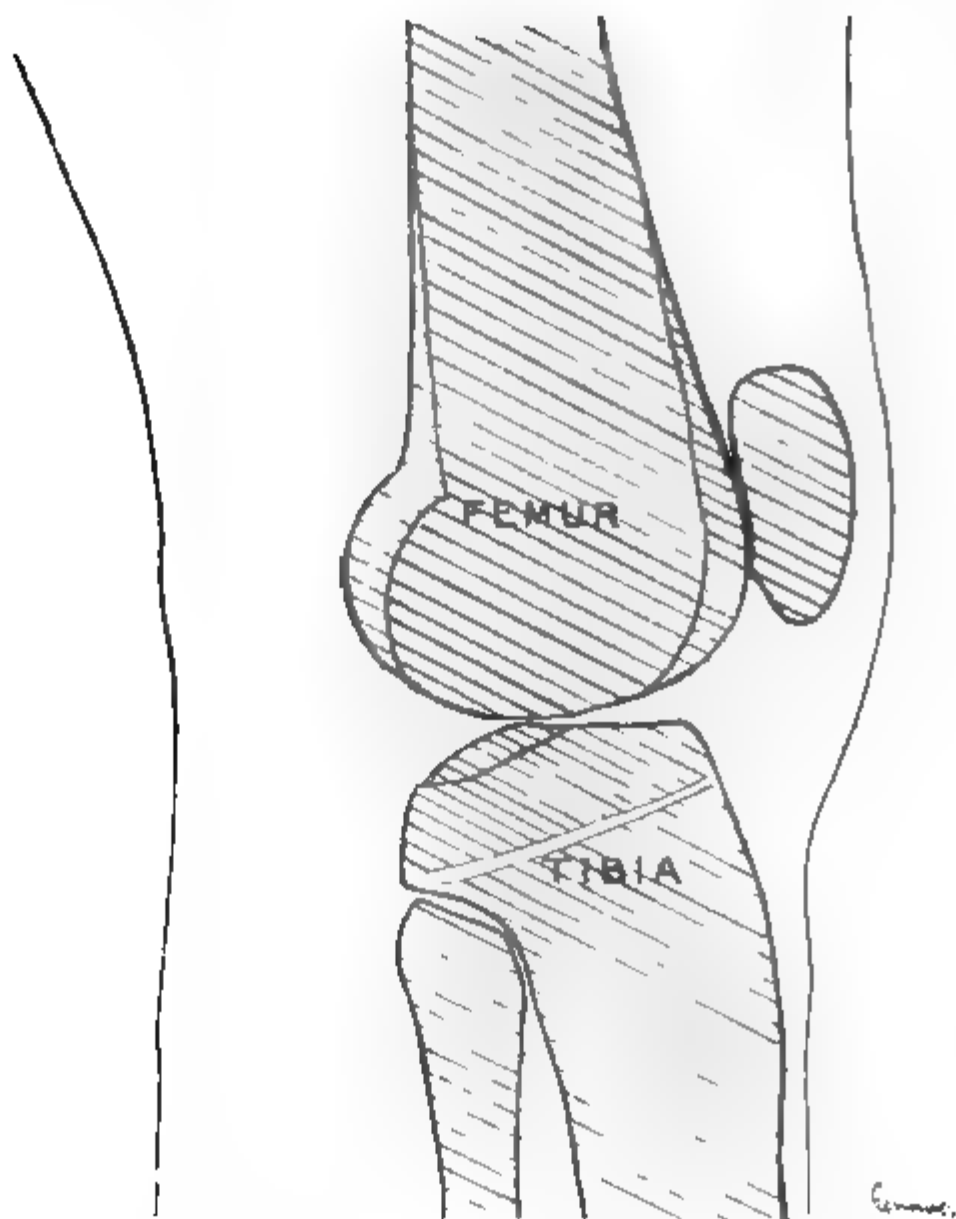
Skiagraph from Case 7, obtained ten years after the accident. The epiphysial line of the femur has become obliterated.

pletely fused with the diaphysis without the least sign of displacement. As the upper epiphysial line of the tibia has not yet disappeared it is certain that the accident has resulted in somewhat premature fusion. This is confirmed by the fact that there is one inch shortening of the right lower limb, at least three quarters of which is in the femur. This shortening has led to slight scoliosis and obliquity of the pelvis; the right thigh is half an inch smaller

in circumference than the left. Flexion and extension of the knee
are perfect, and he walks and runs well. He is engaged as a
painter, and does much kneeling. Since the accident, ten years
ago, he has twice had synovitis in that knee-joint.

CASE 8.—Separation at age of 12; examined at age of 17.
E. H— was admitted into the London Hospital for complete dis-

FIG. 11.
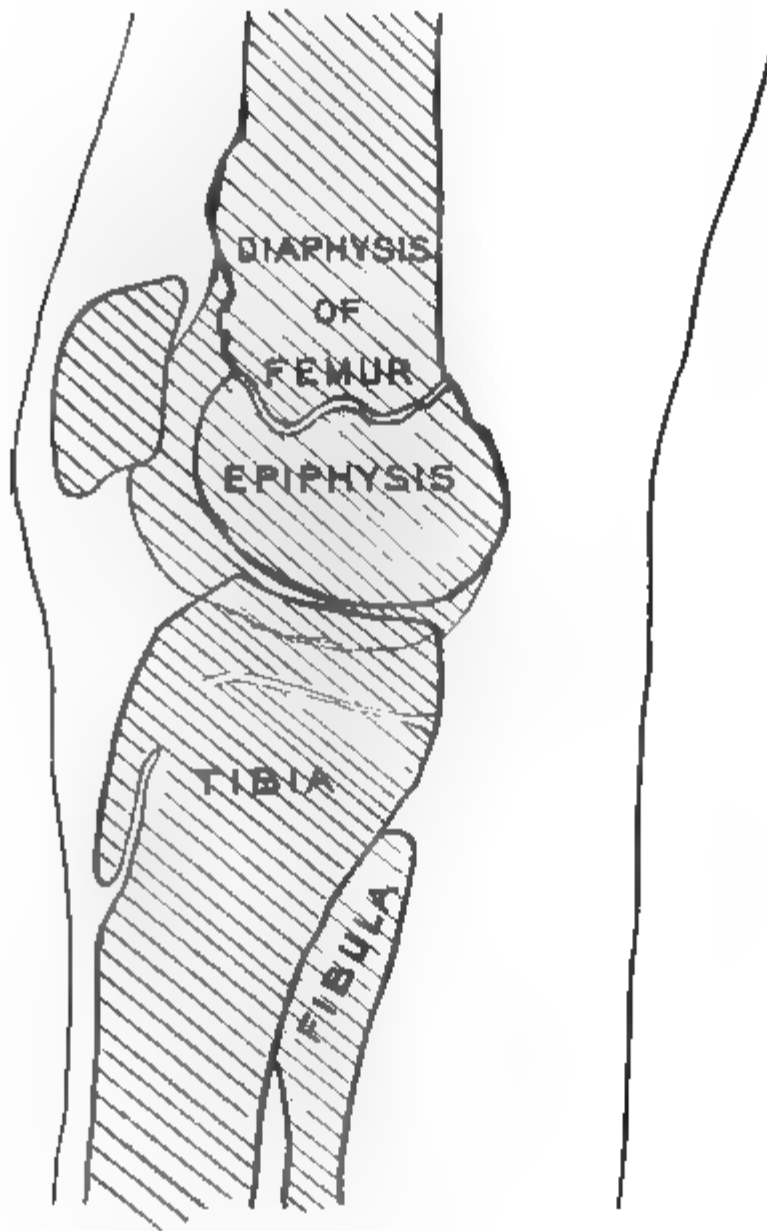
Skiagraph from Case 8, obtained six years after the accident. There
is some irregularity of the diaphysial end.

placement of the lower epiphysis of the femur, which was reduced
under an anæsthetic, and the limb put up on a Macintyre splint,

bent to a right angle. The accident had occurred when he was carrying three boys on his back, the left leg giving way at the knee. This was on the 9th of July, 1892. There was synovitis requiring the use of an ice-bag for a long time. Two months after the accident the limb was gradually extended, and passive motion employed; this caused a return of the swelling; there was then half an inch shortening of the limb. By October 6th he could walk a little, using crutches; but on the 17th the knee was still very much bent, and he limped. He was in the hospital in all seventeen weeks. It seems certain from the shortening that the displacement was not wholly corrected, and it is probable that if the treatment by full flexion had been carried out the duration of the treatment would have been considerably shortened. He was examined again February, 1898—nearly six years later. The skiagraph (Fig. 11) shows the epiphysis in good position, but some new bone in front of the lower end of the diaphysis. There is an inch shortening in the limb. Extension is perfect, but he can only bend the knee a little beyond a right angle. The articular surface of the femur appears to be widened and twisted outwards; there is some thickening behind the internal condyle, and the shortening has led to some scoliosis and obliquity of the pelvis.

CASE 9.—*Outward displacement of the epiphysis in 1889; patient examined nine years later.*—The patient, a boy aged 8, when running behind a hansom, caught his right leg in the wheel, and was dragged fifty yards. On admission the leg was abducted at the knee to an angle of 30° or 40°. Under an anæsthetic it was found that the lower epiphysis of the femur was displaced outwards, and a soft kind of crepitus was felt on reduction. A back splint was applied, with the knee flexed. A fortnight later there was still swelling and slight deformity. After four weeks a Croft's splint was applied. When examined at the age of eighteen there was no shortening, and the limb was in every respect as good as the other one. The skiagram showed no deformity.

CASE 10.—George H—, aged 6, was treated in the London Hospital for seven weeks for separation of the lower epiphysis of the right femur, but the notes are unfortunately so deficient that it is enough merely to state that twelve years later there was no shortening, and the limb was in every respect as good as the other one.

In the following two cases, which occurred in 1888 and 1890, it has been impossible to trace the patient, but they are of sufficient intrinsic interest to be included.

CASE 11.—Alfred F—, February 17th to April 18th, 1888. · In hospital sixty-eight days.

Left femur fractured; right lower epiphysis of femur separated; left humerus, simple fracture at the middle; sternum fractured in lower third.

History.—Caught his coat in engine band; brought in stunned. (We omit the notes which do not relate to the epiphysial separation.)

Right femur.—Knee-joint kept somewhat flexed on a Macintyre splint; considerable effusion into the joint, but less than there was; patella floats in its normal position, but can readily be pressed backwards so as to rest on the front of the condyles. It is impossible to say whether there is any shortening, owing to the fracture of the other femur and the different positions of the thighs, but at present the measurement from the ant. sup. spine to the lower border of the patella is the same on both sides. There is considerable bruising on the back of the knee on the outer side, just over as well as below a marked projection of bone which appears to be connected with the shaft of the femur, possibly being the lower end of the diaphysis; at present it is only just beneath the skin. The axis of the limb does not seem to be much altered, but following down the shaft of the femur it appears to incline backwards, and on pressing gently on it movement is communicated to the projecting fragment. Pulsation can be felt in the post. tibial artery.

March 1st.—Splints removed from right leg. Abrasion on outer. side painted with collodion and iodoform paste.

22nd.—Mr. McCarthy, now that he is able to examine both legs and compare them, thinks there is no doubt that the process of bone to be felt on the right side, which at one time was taken for the separated head of the fibula, is really a process connected with the external condyle of femur, proving the diagnosis of separation of the lower epiphysis correct.

April 18th.—Patient was discharged. He can walk about on crutches with comparative ease, and uses the left leg, the femur of which was fractured; but the right leg is more or less useless; the right knee is much larger than the left, and the piece of bone mentioned above can be plainly felt.

CASE 12.—Arnold S—, aged 10. In hospital forty-four days— September 20th to November 3rd, 1890.

History.—Accident on the morning of September 20th. He was climbing on to the back of a trolley, with his feet on the axle, when the trolley moved forward. His right foot was caught in the spokes of the wheel, and his leg was pulled very violently, so that he fell to the ground.

Present condition.—One inch shortening on the right side, measured from ant. sup. spine to the lower border of the patella. The leg is bowed outwards; the diaphysis projects outwards, and its lower end is felt as a rounded mass about one inch above the head of the fibula. The epiphysis can be distinctly felt on its inner side. There is a well-marked concavity on the inner aspect and convexity on the outer.

Treatment.—The leg was put up on a Macintyre splint, with the knee flexed and an ice-bag applied after an anæsthetic had been given, and reduction effected with the knee flexed without material difficulty.

October 27th.—Patient has been kept in bed, the leg having been on a Macintyre splint, and he has progressed favourably.

28th.—Good movement of knee. Each leg measures 26 inches, and there is perhaps slight displacement of the diaphysis backwards.

CASE 13.—An additional opportunity of testing the method advocated has been afforded by a patient aged 10, in the London Hospital, under the care of Mr. Mansell Moullin. Whilst riding behind a cab the boy caught his left foot in the wheel and was swung round, sustaining concussion and a complete separation of the lower epiphysis of the left femur. The diaphysis could be felt in the lower and outer part of the popliteal space nearly as low as the head of the fibula. There was shortening of 1½ inches and much effusion of blood into the knee.

Treatment.—Extension was made on the leg, whilst the lower end of the diaphysis was drawn forwards so as to flex the knee-joint; when the heel touched the buttock it was fixed in that position by straps and a bandage. During the next ten days the limb was kept in this position, resting on its outer side on a pillow, and *there was no tendency to displacement, although the boy was restless and delirious for several days* as the result of the concussion.

An ice-bag was applied to the knee-joint. At the end of the ten days the limb was taken down and placed in a Macintyre splint flexed to 130°.

At the end of four weeks the limb had been gradually extended; two weeks later the knee could be fully flexed and almost completely extended. After being up in gum and chalk for some further period slight stiffness remained, but promised to wholly clear off under massage, &c.

The two limbs were exactly the same length, and the left knee was only one third of an inch greater in circumference than the right. This case was throughout under the observation of Mr. Barnard, and we are indebted to Mr. Mansell-Moullin for permission

to test the treatment and to record the result. Owing to the extreme displacement and the complication of the head injury, it furnished a specially good test of the full flexion method, and the result left nothing to be desired.

CONCLUSIONS.

1. That separation of the lower epiphysis of the femur is a very serious injury, attended when compound by a high mortality.

2. That in the extended position of the knee even with an anæsthetic reduction of the fragment is often very difficult.

3. That when treated by extension and a long Liston's splint it is almost impossible to keep the fragments in position, and with a Macintyre splint it is sometimes difficult.

4. Nevertheless the ultimate result, in most cases that recover at all, is very good. The articular surface of the femur gradually grows into a useful position.

5. That with the method advocated by us reduction is always easy, the time of treatment is short, and it is the rule to obtain perfect movement in the knee without shortening or deformity of the leg.

(For report of the discussion on this paper, see ' Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. xi, p. 29.)

AORTIC ANEURYSM

AS A CAUSE OF HYPERTROPHY OF THE LEFT VENTRICLE

BY

JAMES CALVERT, M.D.

DEMONSTRATOR OF MORBID ANATOMY IN ST. BARTHOLOMEW'S HOSPITAL

SOME authorities state that aortic aneurysm is a cause of hypertrophy of the left ventricle, and others are equally positive that it is not. This conflict of opinion is embarrassing to the teacher and confusing to the student.

I have therefore thought it worth while to see what light the post-mortem records of St. Bartholomew's Hospital throw upon the subject, and my examination of the " Record of Complete Cases " has been made a comparatively easy task by Dr. Oswald Browne's recent dissertation for the degree of M.D. at Cambridge, entitled " Aneurysms of the Aorta."[1]

Dr. Browne's thesis is based " upon an analysis of all cases of aneurysm of the aorta in any of its parts dying in St. Bartholomew's Hospital during the last thirty years, upon whom an examination was made after death and its results carefully recorded." .

(a) Of the ascending part of the arch he has analysed 58 cases :

[1] 'Aneurysms of the Aorta,' by Oswald A. Browne, M.A., M.D. H. K. Lewis, London, 1897.

In 4 of these the condition of the heart was not recorded in the post-mortem notes.

In 1 case the heart is described in the post-mortem notes as " fatty."

In 27 cases there was no hypertrophy of the left ventricle.

In 26 cases there was hypertrophy of the left ventricle.

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58

(β) Of the transverse part of the arch he has analysed 35 cases :

In 1 of these the description of the heart in the post-mortem notes is " some general dilatation."

In 2 cases the heart is described as " fatty."

In 19 cases there was no hypertrophy of the left ventricle.

In 13 cases there was hypertrophy of the left ventricle.

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35

(γ) Of aneurysms affecting both the ascending and transverse parts of the arch he has analysed 19 cases :

In 12 cases there was no hypertrophy of the left ventricle.

In 7 cases there was hypertrophy of the left ventricle.

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19

(δ) Of the descending part of the arch he has analysed 21 cases :

In 1 case the condition of the heart was not recorded in the post-mortem notes.

In 10 cases there was no hypertrophy of the left ventricle.

In 10 cases there was hypertrophy of the left ventricle.

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21

The particulars of these cases with hypertrophy of the left ventricle, copied from Dr. Browne's tables and retaining his numbering, are as follows :

TABLE I.—Aneurysms of the First or Ascending Portion of the Arch.

No.	Reference.	Sex.	Age.	Part of aorta affected.	Direction and effects.	State of heart.	State of valves.	State of aorta.	Cause of death.	Observer.
1	I, 201	M.	40	1 inch above aortic valves; arch gradually dilated into a fusiform aneurysm; hence an opening into a sacculus abutting on and almost occluding vena cava superior (A smaller sacculus anteriorly)	Pressure on vena cava superior	Some hypertrophy of left ventricle	Aortic valve thickened	Calcification in wall of sacculus; slight puckering elsewhere	Œdema of lungs	Dr. Church.
2	I, 244	M.	40	Greatly dilated ascending aorta, with large sacculated aneurysm in connection with right lateral wall, springing 2 inches above aortic valve. A second aneurysm from anterior and left wall just below origin of innominate artery. A third, formed by dilatation of aortic wall at commencement of descending portion. Two or three further aneurysmal dilatations of abdominal aorta	Occupying anterior mediastinum and right pleural cavity. Causing erosion of left side of sternum, 3rd costal cartilage and rib, and absorption of intercostal muscles. Slightly eroding vertebræ	Slightly hypertrophied; much dilatation of left ventricle	Healthy	Slight atheroma at commencement	Increasing dyspnœa; collapse and softening of right lung	Dr. Church.

N².	Reference	Sex & Age	Part of aorta affected	Direction and effects	State of heart	State of valves	State of aorta	Cause of death	Observer
5	II, 127	M. 56	Right side of ascending portion	Tumour in anterior mediastinum immediately above pericardium, rupturing enormously through there into right pleura	Left ventricle hypertrophied	Aortic valve thickened and incompetent	Ascending and transverse portions greatly dilated	Rupture into right pleural cavity	Dr. Ghee
7	II, 260	M. 36	Right side of ascending portion	Occupying anterior mediastinum; heart depressed; lungs pushed to right and left; vena cava superior and venæ innominatæ much compressed; some pressure on right pulmonary artery	Some hypertrophy of left ventricle	Healthy	Slight atheroma throughout	? Large serous effusion in both pleural cavities	Dr. Ghee
8	II, 303	M. 32	A slit immediately above aneurysmal cavity led directly into vena cava superior	Opening near heart into vena cava superior valve	Some hypertrophy of left ventricle	Slightly atheromatous	Highly atheromatous	—	Dr. Wickham Legg.
11	III, 818	M. 57	Whole arch greatly dilated to 1 inch below left subclavian just above one semilunar valve on right anterior aspect of aorta is a ragged opening into pericardium; large vessels not implicated	Trachea much compressed by small pouch from aneurysm; one cartilage eroded; left vena innominata crosses aneurysm	Wall of left ventricle thickened	Healthy	Descending aorta full of bony plates	Rupture into pericardium	Dr. Ghee
12	IV, 114	M. 37	Sacculus behind posterior semilunar valve	—	Great general hypertrophy	Aortic valves incompetent	Slightly dilated	—	Dr. Norman Moore.

No.	Ref.	Sex	Age	Location	Description	Left ventricle	Aortic valve	Aorta	Remarks	Reporter
15	V, 97	F.	53	Right side of aorta immediately above valves	—	Left ventricle hypertrophied	Aortic valve thickened; mitral atheromatous	Fairly healthy	—	Dr. Wickham Legg.
16	V, 173	F.	40	Immediately above valves	Bulging from right side of this presses much on superior vena cava; a bulging from left side presses on pulmonary artery	Much hypertrophy of left ventricle	Aortic and mitral highly atheromatous, incompetent	Atheromatous throughout	Extreme dyspnoea	Dr. Wickham Legg.
17	V, 275	M.	42	Ascending part of arch 1 inch above valves	Extending forwards and to right, pressing on left side of pulmonary artery, also upon inferior vena cava and superior vena cava	Slight hypertrophy of left ventricle	Healthy	Slight atheroma	—	Dr. Norman Moore.
19	V, 345	M.	37	Aorta dilated immediately above valves; hence lead two sacs, one to right, one from back part of aorta, also bulging to right	This pressed on superior vena cava and ruptured into pericardium	Left ventricle scarcely hypertrophied	Healthy	Some atheroma	Rupture into pericardium	Dr. Wickham Legg.
23	VII, 87	M.	44	Aorta greatly dilated from immediately above valves to origin of innominate; hence a small aneurysmal sac	Tumour projected at upper border of right axilla	Great hypertrophy of left ventricle	Aortic valves incompetent	Highly atheromatous to left subclavian; slightly so	Infarction in both lower pulmonary lobes	Dr. Norman Moore.

Refer-ence	Sex, age	Part of aorta affected	Direction and effects	State of heart	State of valves	State of aorta	Cause of death	Observer
23 VII, 145	M. 45	Large aneurysm above coronary arteries and below innominate artery	Enormous tumour in middle of chest and on right side; right lung firmly adherent and forming wall of cavity; second, third, and fourth right ribs much eroded, with much of upper part of sternum	"Of little more than normal size"	Healthy	Highly athero-matous	? Large double pleural effusion	Dr. Norman Moore
25 VII, 259	M. 27	Small aneurysm between orifices of coronary arteries	Pointing towards pulmonary artery and pressing upon it just above valves	Left walls hyper-trophied; weight 3 lbs.	Aortic valves thick-ened, incom-petent	Arch highly athero-matous	? Oedema of lungs	Dr. Ormerod.
26 VII, 293	M. 54	Large aneurysm of ante-rior part of ascending por-tion, with fusiform exten-sion up innominate artery; a small additional sac be-yond left subclavian	Tumour adherent to chest wall from third left rib upwards; left lung re-tracted and adherent to back of tumour; calibre of main pulmonary artery and of its left branch nar-rowed; left vagus nerve also compressed	Hyper-trophy of left ventricle	Aortic valves thick-ened	Highly athero-matous	—	Dr. Norman Moore.
28 VIII, 341	F. 15	Between attachment of peri-cardium and origin of in-nominate artery; great vessels natural	Tumour covered ascending aorta	Wall of left ventricle thickened	Mitral stenosis; aortic cusps adherent	Healthy	—	Dr. Ormerod.
29 X, 104	M. 40	Aorta dilated into aneu-rysmal sac just above valves	Bulk of sac lay behind and to left of vessel; commu-nication with pulmonary artery; no external tumour	Heart very flabby; weight 14 oz.	No note	Not else-where athe-romatous	Rupture into pulmonary artery	Dr. Tooth.

No.	Case	Sex	Age			Weight	Valves	Aorta	Symptom	Reporter
30	XI, 47	M.	69	Small bulging immediately above valves; fusiform aneurysm 2 inches above bifurcation of aorta	—	Weight 18 oz.	Healthy	Athero-matous	—	Dr. Norman Moore.
33	XII, 49	M.	31	1½ inches above valves	Forwards, penetrating sternum; manubrium eroded; external tumour over sternum at level of second rib	Slight hypertrophy of both ventricles	Healthy	Much atheroma for 1½ in. above valves, not elsewhere	Urgent dyspnœa	Dr. Norman Moore.
35	XII, 181	F.	46	Immediately above aortic valves	Wholly within pericardium	Some general hypertrophy, especially of left ventricle	Aortic valves thickened	Highly athero-matous	—	Dr. Norman Moore.
36	XII, 203	F.	35	Immediately above aortic valves, not involving sinuses of Valsalva	Backwards and to right; right bronchus slightly flattened; no external tumour	General hypertrophy and dilatation	Healthy	Athero-matous	? Right pleural effusion; thrombosis of right brachial and both femoral veins	Dr. Ormerod.
46	XIX, 297	M	—	Just above aortic valves	A secondary pouch pressed upon pulmonary artery just above valves	Dilated, flabby; weight 24 oz.	Incompetent	Athero-matous	Increasing dyspnœa	Dr. Ormerod.
47	XX, 330	M.	53	Large aneurysm arose from anterior wall of ascending arch	Sac adherent to right lung and to first and second right costal cartilages close to sternum; no erosion	Hypertrophy of left ventricle	Aortic valves thickened	Athero-matous	Rupture into right pleural cavity	Dr. Tooth.
48	XX, 366	M.	50	Small saccular aneurysm about 2 inches above valves	No external tumour	Much hypertrophy	Healthy	Highly atheroma-tous	Œdema of lungs	Dr. Tooth.

TABLE II.—*Aneurysms of the Second or Transverse Part of the Arch.*

No.	Reference	Sex	Age	Part of aorta affected.	Direction and effects.	State of heart.	State of valves.	State of aorta.	Cause of death.	Observer.
50	XXI, 75	M	48	Aneurysm of ascending part of arch	Communicated by two small openings with pulmonary artery; no external tumour	Left ventricle hypertrophied	Aortic valve thickened	Atheromatous	——	Dr. Tooth.
52	XXI, 293	M	29	Aneurysmal dilatation of ascending arch	Principally to right; no visible tumour	Hypertrophy and dilatation of left ventricle	Aortic valve incompetent; cusps much diseased	Atheroma of ascending arch	Heart failure	Dr. Tooth.
6	I, 286	M	44	Aorta immediately above valves dilated into large globular sacculus with atheromatous walls; 3 inches above valves oval opening leads into pouch, of which walls formed by connective tissue surrounding this part of arch	Anterior wall of tumour adherent to depression in sternum at junction of 3rd left costal cartilage; some compression of vena cava superior; parts of wall firmly adherent to pericardium; right lung collapsed	Left ventricle hypertrophied	Healthy	Atheromatous	? Œdema of lungs	Dr. Church.
7	II, 178	F.	60	Rupture through inner and middle coats at origin of innominate artery; external coat quite separated from middle coat, blood-clot intervening	Rupture into pericardium at right side of aorta, just where arising from heart	Left ventricle somewhat hypertrophied	No note	Atheromatous	Rupture into pericardium	Dr. Gee.

No.	Case	Sex	Age	Description	Left ventricle	Aortic valves	Aorta	Tracheotomy performed for dyspnoea	Reporter
9	III, 48	M.	89	Sacculus formed by great distension of posterior wall of innominate artery at its origin — Great pressure on anterior wall of trachea; no erosion; left recurrent nerve much flattened	Left ventricle much hypertrophied	Aortic valves thickened, incompetent	Highly atheromatous	Tracheotomy performed for dyspnoea	Dr. Gee.
11	IV, 386	M.	36	Middle portion of arch converted into aneurysmal sacculus; innominate artery had disappeared; right carotid and subclavian spring directly from sacculus — Tumour rises high in neck; presses behind on trachea, which in three spots is ready to burst; left innominate vein obliterated where it crosses sac	Left ventricle much hypertrophied	Fine granulations on aortic valve	Arch dilated and highly atheromatous	Increasing tracheal compression	Dr. Wickham Legg.
14	V, 137	F	36	Aneurysm of transverse and descending portions; a small sacculus projects from its inner side — Sac and main aneurysm both pressed on left bronchus and on trachea just above bifurcation; wall of trachea eroded; no external tumour	Natural	Healthy	No note	Asphyxia	Dr. Norman Moore.
15	VI, 24	F	53	Aorta natural to left carotid, there opening into trifid sac — One sac eroded bodies of 3rd and 4th dorsal vertebræ; another opened into bronchial tube 1¼ inches below bifurcation of trachea; the 3rd passed into main stem of pulmonary artery	Left ventricle natural	Healthy	Lower part atheromatous	—	Dr. Wickham Legg.
19	IX, 286	M	41	Aneurysm extended from origin of innominate to left subclavian artery — Projecting mainly forwards through 1st left intercostal space and sternum, and barely covered by thin layer of sternum and of pectoral muscle; contraction of left lung	Slight general hypertrophy	Healthy	Atheromatous throughout	—	Dr. Norman Moore.
21	X, 125	M.	45	First aneurysm projected from arch anteriorly and slightly to right, just below origin of innominate artery; a second projected backwards from arch below, and above origin of — Bulging inwards of anterior wall of trachea just above bifurcation; pulsation in 2nd left interspace; no external tumour	Slight hypertrophy of left ventricle	Healthy	Great general dilatation, with much atheroma from valves to junction	Sudden dyspnoea	Dr. Norman Moore.

No.	Reference.	Sex	Age	Part of aorta affected.	Direction and effects.	State of heart.	State of valves.	State of aorta.	Cause of death.	Observer.
22	X, 353	M.	66	Whole of transverse and upper part of descending arch dilated into large aneurysmal sac	Part of left lung adherent to sac; left upper lobe full of blood; no external tumour	Weight 20 oz.	Healthy	Highly atheromatous	Death sudden	Dr. Tooth.
24	XI, 168	M.	50	Wide opening into large aneurysmal sac 2¼ inches above valves	Projecting *forwards* in 2nd right intercostal space and there penetrating the muscle, and *upwards* above episternal notch, and adherent to and pressing *forwards* upper part of sternum; *backwards* and to right it compressed right lung, which was adherent to it; sternum eroded from 2nd right costal cartilage to left left costal cartilage, especially at right side	Heart slightly hypertrophied	Healthy	First 2 inches atheromatous	Great oedema of arytæno-epiglottidean folds, quite closing orifice of larynx	Dr. Norman Moore.
26	XIII, 96	M	41	Sacculated aneurysm from front of transverse part of arch	Perforation of second bone of sternum on either side, central portion intact; external tumour to left of sternum, opposite 2nd, 3rd, and 4th left costal cartilages, also to right of sternum, at level of 2nd costal cartilage	Slight hypertrophy of left ventricle	Not noted	Very atheromatous	External rupture	Dr. Norman Moore.
32	XXI, 181	M.	58	Large aneurysm of transverse part, involving also large part of descending portion	Aneurysm adherent to upper part of left lung; bodies of dorsal vertebræ—5, 6, 7, 8—much eroded	Weight 12 oz.	Healthy	Highly atheromatous	Rupture into left pleural cavity	Dr. Tooth.

34	XXIII, 201	M.	30	Saccular aneurysm arose more to left than right of trachea; no external tumour posteriorly, close to, but not involving, innominate artery. Another small aneurysm 2 inches above valves	Enormous hypertrophy and dilatation of left ventricle	Aortic valves incompetent	Very atheromatous	Recurring attacks of dyspnoea	Dr. Garrod.
35	XXIII, 312	F.	44	From 1¼ inches beyond origin of innominate to just beyond left subclavian artery	Had almost perforated trachea in two places; no external tumour	Some hypertrophy of left ventricle	Aortic valves competent	Athero- matous	Asphyxia	Dr. Calvert.

TABLE III.—*Aneurysms of the Ascending and Transverse Portions of the Arch.*

3	VII, 273	M	39	Aneurysm above valves and extending 1 inch beyond origin of left subclavian	Cavity immediately extended chiefly upwards and backwards and against trachea; trachea perforated just above bifurcation	Slight hyper- trophy	Healthy	Highly athero- matous	—	Dr. Norman Moore.
4	VIII, 292	M.	46	Aneurysm included whole of arch as far as left subclavian, extending anteriorly and posteriorly; funnel-shaped dilatation of left subclavian for 1½ inches from origin	Projection of tumour between 1st and 3rd right ribs; 2nd right costal cartilage and part of sternum eroded; trachea much compressed just above bifurcation, two rings partially eroded; bodies of the two upper dorsal vertebræ eroded on right side	Both ventricles slightly hyper- trophied	Healthy	Athero- matous	Increasing dyspnœa	Dr. Norman Moore.
6	XI, 267	M.	48	From just above aortic valves to origin of left subclavian	Backwards towards apex of right lung, which was collapsed and adherent to aneurysm; here rupture; tumour projected from 3rd right interspace to clavicle; 2nd rib much thinned	Slight hypertro- phy of left ventricle	Healthy	Athero- matous	Rupture into right pleural cavity	Dr. Norman Moore.

No.	Reference.	Sex.	Age.	Part of aorta affected.	Direction and effects.	State of heart.	State of valves.	State of aorta.	Cause of death.	Observer.
9	XVI, 81	M.	31	Aneurysm from posterior part of ascending and transverse portions of arch; great vessels not involved	Aneurysm seated on front of trachea, which was not compressed; rupture into tissues in front of trachea; left recurrent laryngeal nerve involved; no external tumour	Left ventricle hypertrophied	Aortic valves thickened	Atheromatous	Tracheotomy performed for urgent dyspnœa	Dr. Ormerod.
11	XVIII, 221	M.	38	Ascending and transverse portions of arch; large vessels arose from upper part of sac	2nd right costal cartilage eroded	Left ventricle hypertrophied	Aortic valves incompetent	Atheromatous throughout	Septic pneumonia	Dr. Ormerod.
12	XIX, 120	M.	60	Aneurysm commenced within pericardium, involved whole of arch, terminating just beyond origin of left subclavian; orifices of great vessels normal	None noted	Left ventricle hypertrophied; right side dilated	Healthy	Highly atheromatous	? Right pleural effusion	Dr. Ormerod.
15	XXII, 225	F.	49	Large aneurysm of ascending and transverse portions of arch; innominate and left carotid arteries emerged from sac; left subclavian free	Perforated right border of sternum, and second and third right costal cartilages	Weight 15 oz.; fatty	Healthy	Very atheromatous throughout	Asphyxia	Dr. Garrod.

TABLE IV.—*Aneurysms of the Third or Descending Part of the Arch.*

No.	Reference.	Sex.	Age.	Part of aorta affected.	Direction and effects.	State of heart.	State of valves.	State of aorta.	Cause of death.	Observer.
2	IV, 133	M.	34	Just below origin of left subclavian artery	Pouch lying between aorta and apex of left lung, which formed its anterior	Left side of heart hyper-	Healthy	Highly atheromatous	Phthisis; cirrhosis hepatis	Dr. Wickham Legg.

6	VII, 277	M.	42	Just beyond origin of left subclavian posterior surface of aorta bulged into an aneurysm; a slighter bulging on its opposite wall	Sac bursting downwards had dissected mucous from muscular coat of œsophagus as far as upper surface of diaphragm, where blood had flowed through a small orifice into left pleural cavity	Left ventricle slightly hypertrophied	Healthy	Highly atheromatous above valves	Rupture into left pleural cavity	Dr. Norman Moore.
7	VIII, 282	F.	43	Aneurysm extended for 2 inches from origin of left subclavian artery	Upper part of left lung forms part of wall of aneurysm, which had ruptured into left pleura	Left ventricle hypertrophied	Healthy	Highly atheromatous	Rupture into left pleural cavity	Dr. Norman Moore.
8	VIII, 309	M.	43	At commencement of descending part just below origin of left subclavian artery	Extending chiefly backwards; left lung firmly adherent to and forming part of aneurysmal wall, as also the much eroded bodies of 4th, 5th, and 6th dorsal vertebræ; rupture through lung into left pleura	Heart "not greatly hypertrophied"	Healthy	Highly atheromatous below aneurysm	Rupture into left pleural cavity	Dr. Norman Moore.
9	X, 365	M.	38	Saccular aneurysm of descending portion	Adherent to inner aspect of apex of left lung; 4th, 5th, 6th, and 7th left ribs eroded; rupture at lower part, close to vertebræ, into left pleural cavity	Weight 13 oz.	Healthy	Some atheroma of arch	Rupture into left pleural cavity	Dr. Tooth.
11	XIII, 71	M.	55	Aneurysm of third part of arch and upper part of descending thoracic aorta	Wall of sac in part formed by tissue of left lung; 3rd and 4th dorsal vertebræ eroded on left side; rupture into left pleura	Slight hypertrophy of left ventricle	Two aortic cusps adherent	No calcification below aneurysm	Rupture into left pleural cavity	Dr. Norman Moore.
15	XX, 366	M.	52	Large fusiform aneurysm of half of transverse and whole of descending part of arch	Aneurysm adherent to left lung; rupture into left pleural cavity	Some hypertrophy of left ventricle	Healthy	Very atheromatous	Rupture into left pleural cavity	Dr. Tooth.

No.	Reference	Sex	Age	Part of aorta affected	Direction and extent	State of heart	State of valves	State of aorta	Cause of death	Observer
16	XXI, 288	M.	40	Aneurysm of descending part of arch	As arch passed over root of left lung, small loculus compressed left pulmonary artery; lower part of aneurysm adherent to oesophagus at level of bifurcation of trachea; here rupture into oesophagus	No marked hypertrophy of left ventricle	Healthy	Slight atheroma	Rupture into oesophagus	Dr. Tooth.
17	XXII, 884	M.	67	Small aneurysm on right side of lower part of descending arch	Sac adherent to right lung; rupture into right lung a little above right bronchus	Much hypertrophy of left ventricle	Aortic valve thickened	General atheroma	Rupture into right lung	Dr. Tooth.
20	XXIII, 288	M.	35	Descending part of arch	Main sac eroded on left side of bodies of 5th, 6th, and 7th dorsal vertebrae. A smaller (false) aneurysm had compressed and finally ruptured into left bronchus; this had also compressed and flattened left pulmonary artery	Much hypertrophy, especially of left ventricle	Aortic valves very incompetent	Extreme atheroma throughout	Rupture into left bronchus	Dr. Garrod.

In the ascending part of the arch we have 27 cases without hypertrophy, 26 cases with hypertrophy. But with regard to these 26 cases with hypertrophy,—

In Nos. 12, 16, 23, 25, 46, 52 . . . . . 6 cases,
    the aortic valves were incompetent.
In Nos. 17, 29, 35 . . . . 3 cases,
    there was adherent pericardium.
In Nos. 8, 24, 26 . . . . 3 cases,
    the aorta is described as highly atheromatous.
And on referring to the post-mortem records I find
    that in Nos. 1, 2, 5, 15, 28, 30, 36, 47, 48 . . 9 cases,
    the kidneys were granular.
And on referring to the clinical notes of the case I
    find that in No. 50 . . . . . . 1 case,
    there was well-marked aortic regurgitation.   —
                                22 cases.

(In Nos. 8 and 29 the condition of the kidney is not recorded
    in the post-mortem notes.)

Thus the hypertrophy in 22 of the 26 cases can be perfectly explained without the aid of aortic aneurysm. Four cases remain—Nos. 7, 11, 19, 33:

But in No. 19 the heart is described as "scarcely hypertrophied,"
    therefore it may be left out.
In No. 7.—Some hypertrophy of left ventricle; some atheroma
    of aorta; kidney natural.
In No. 11.—"Wall of left ventricle rather thick," "kidneys
    indurated as in cardiac disease," are the expressions used in
    the post-mortem records. "Descending aorta full of bony
    plates," so that no doubt there was atheroma elsewhere in
    the aorta.
In No. 33.—Slight hypertrophy of both ventricles; kidney
    natural; much atheroma for 1¼ inches above the aortic
    valves.

In each of these 3 cases the hypertrophy is slight, and may probably be accounted for by the condition of the arteries.

In the transverse part of the arch we have 19 cases without hypertrophy, 13 cases with hypertrophy. But with regard to these 13 cases with hypertrophy,—

In Nos. 9, 34 .   .   .   . 2 cases,
    the aortic valves were incompetent.
In No. 11 .   .   .   . 1 case,
    the aorta is described as highly atheromatous.
And on referring to the post-mortem records I find
    that in Nos. 6, 7, 16, 22, 26, 32, 35 .   .   . 7 cases,
    the kidneys were granular.   —
                                               10 cases.

Thus the hypertrophy in 10 of the 13 cases can be satisfactorily accounted for without the aid of aortic aneurysm.

With regard to the 3 remaining cases,—

    In No. 19.—The kidneys were "large and firm;" the aorta atheromatous throughout; slight general hypertrophy.

    In No. 21.—Capsule of kidney slightly adherent; much atheroma of first and second parts of the arch; slight hypertrophy.

    In No. 24.—Kidneys congested; first 2 inches of aorta atheromatous; slight hypertrophy.

In each of these cases the hypertrophy is slight, and may probably be accounted for by the condition of the arteries.

Of aneurysms affecting both the ascending and transverse parts of the arch we have 12 cases without hypertrophy, 7 cases with hypertrophy. But with regard to these 7 cases with hypertrophy,—

In No. 11 .   .   .   . 1 case,
    the aortic valves were incompetent.
In No. 3 .   .   .   . 1 case,
    the aorta is described as highly atheromatous.
And on referring to the post-mortem records I find
    in Nos. 12, 15 .   .   .   . 2 cases,
    the kidneys were granular.
And on referring to the clinical notes I find in No. 9 1 case,
    there was well-marked aortic regurgitation.   —
                                            5 cases.

Thus the hypertrophy in 5 cases of the 7 can be satisfactorily explained without the aid of aortic aneurysm.

With regard to the 2 remaining cases,—

In No. 4.—Slight hypertrophy of both ventricles; aorta athe-
romatous; kidney, two solitary cysts, otherwise natural.
In No. 6.—Slight hypertrophy of left ventricle; aorta athero-
matous; kidney natural.

Here, as before, the hypertrophy is slight, and is pro-
bably due to the condition of the arteries.

In aneurisms of the descending part of the arch we
have 10 cases without hypertrophy, 10 cases with hyper-
trophy. But with regard to these 10 cases with hyper-
trophy,—

In Nos. 2, 6, 7 . . . . 3 cases,
    the aorta is described as "highly atheromatous."
And on referring to the post-mortem records I find in
    Nos. 8, 11, 15, 16, 17, 20 . . . . . 6 cases,
    the kidneys were granular.   —
                                        9 cases.

Thus the hypertrophy in 9 cases out of the 10 can be
satisfactorily explained without the aid of aortic aneu-
rism.

With regard to the remaining case,—

No. 9.—Heart weighed 13 oz.; some atheroma of arch; kidney
natural.

Finally, then, taking all the cases together, we have
124 cases of aneurism of the arch of the aorta :

In 68 of them there was no hypertrophy of the left
    ventricle.
In 47 of them the hypertrophy could be perfectly
    explained by other causes present.
In 9 of them the hypertrophy could very probably
    be explained by other causes present.

Therefore, so far as the records of St. Bartholomew's
Hospital are concerned, there is no evidence in favour of
aortic aneurism being a cause of hypertrophy of the left
ventricle.

(For report of the discussion on this paper, see ' Proceedings o
the Royal Medical and Chirurgical Society,' Third Series, vol. xi
p. 37.)

A CASE OF AORTIC ANEURYSM

INVOLVEMENT OF SENSORY NERVE-ROOTS; SPONTA NEOUS FRACTURE OF VERTEBRAL COLUMN; LAMINECTOMY; DEATH

BY

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AND

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ASSISTANT SURGEON TO ST. THOMAS'S HOSPITAL

WILLIAM B—, aged 35, seaman. Admitted to St Thomas's Hospital September 14th, 1897; died Octobe 24th, 1897.

Family history.—Father is alive and healthy; th mother died of bronchitis. Nothing points to inherite disease.

Previous history.—Patient had a hard chancre seventeei years ago, which was followed by a rash, but no othe secondary symptoms. He was treated with black wash but took no medicine. Four years later he married; hi wife is quite healthy, and has borne him three health children, but has had one miscarriage. None of the children have apparently shown any sign of inherite syphilis. Until the commencement of the present illnes patient had always enjoyed good health, and had beer quite free from accidents.

Present illness.—This began two years ago, whei

patient first noticed pain in the middle of his back, which came on without any apparent cause. He attended the Seamen's Hospital, Poplar, for it, and was given a liniment. The pain at first was not constant, but recurred some three or four times a week, generally at night. It was of an aching character, and soon extended round his body at the same level. These attacks have become more and more frequent, and for the last four months have been practically continuous. Of late, however, the pain in the back has become quite subordinate to that referred to the lower and anterior part of the thorax. Movement seems to have no effect upon it, though the back has felt stiff ever since the first onset. Four months ago patient spent three weeks in Sydney Hospital ; but deriving no benefit from the treatment, he returned to England. In Sydney and on board ship he had injections of morphia about once a week, and occasional sleeping draughts. During the last four months he has lost at least two stones in weight.

State on admission.—Patient is a powerfully built man, but thin, and with a well-marked expression of pain on his face. He complains in a wailing voice of intense pain in the upper abdomen and back. He keeps on repeating expressions such as " I can't bear it any longer," and seems constantly on the verge of tears.

Nervous system.—No loss of power or affection of sensation can be detected in the extremities, either upper or lower, and no trophic changes are evident in either skin or muscles. The knee-jerks are present and equal on the two sides, and no cloni can be elicited. The superficial reflexes are equally unaltered. The sphincters act normally, and have always done so. The mental condition is clear, but shows distinct loss of emotional control, which, under the circumstances, is not surprising. In fact, so far as the nervous system is concerned, all signs of disease are limited to a zone of the trunk. The patient refers the pain mainly to the following area, which is of roughly triangular shape, and situated on the anterior right

side of the chest (see Plate I, Fig. 1). Its upper bord₁
is horizontal, and touches the lowest point of the fifth ril
the lower margin starts from the middle line about an inc
below the tip of the ensiform, and after pursuing at fir₁
a horizontal course, runs upwards and outwards to me₁
the upper boundary in the anterior axillary region. Tl
inner vertical margin corresponds to the middle line ₁
the trunk, and unites the inner ends of the lines ju
described. The skin over this area is intensely hype
æsthetic, the patient flinching and often crying out wil
pain when it is gently pinched between the finger ar
thumb. The borders are well defined, the hyperæsthes
suddenly disappearing at the periphery. Tactile sense, ₁
the other hand, is slightly impaired. For example, tʋ
points which are recognised as distinct elsewhere a:
merged into one over the hyperæsthetic area, though ₁
account of the excessive sensitiveness it is difficult
make an altogether satisfactory examination. There is ₁
change in either the vascular or trophic condition of tl
skin.

The corresponding triangle on the left side of the bo₁
is not hyperæsthetic, and at present pain is not referr₁
to that spot, though apparently it has been in the pa₁
The only change which can be detected in its senso₁
functions consists in some blunting of tactile sense, su₁
as has been already described on the opposite side.]
the back there is no zone of hyperæsthesia, and no de₁
tenderness except on pressure over the tenth dors
spine.

Spinal column.—The rigidity of the back at on·
attracts attention ; this is not limited to any one part, b
is more marked in the upper dorsal and cervical regio₁
The stiffness applies both to antero-posterior and
lateral movements. The patient can move about in be
and even walk about the ward, without any increase in t.
pain, and absolutely no effect is produced by firm pressu
applied to the head or shoulders. The back shows a ve
distinct deformity ; the upper half of the dorsal spine h

the normal backward curve visibly exaggerated. This curve terminates rather abruptly, the spinous process which forms the extreme end of the prominence being the eighth dorsal, while the site of maximal projection is occupied by the seventh dorsal. Below the eighth spine there is a marked concavity, the floor of which is formed by the ninth and tenth dorsal spines. The tenth spine is extremely tender. A sponge wrung out of hot water does not produce the least pain at any spot in the column. There does not appear to be any lateral irregularity.

The abdomen is normal in every respect.

Chest.—The cardiac dulness begins above at the fourth left costal cartilage; the apex-beat is in the fifth inter-space, half an inch internal to the left nipple line, while the limit of dulness to the right corresponds with the left margin of the sternum. The sounds are normal both at base and apex, while the rhythm is regular at 72 per minute.

The urine has a specific gravity of 1030, is loaded with urates, and contains neither albumen nor sugar.

On September 21st the tenderness noticed over the tenth vertebral spine was found to have altogether disappeared. Since admission patient had had one or two injections of morphia, and a mixture of cannabis indica with tincture of gelsemium, but without much effect on the pain.

The hyperæsthetic area was less distinct than on admission.

On September 25th the patient, who was very anxious to get up, was allowed to do so. This did not appear to have any effect on the pain one way or the other; but on September 30th the pain grew worse, and he went back to bed, where he thereafter remained. On October 1st specific treatment was commenced in the form of inunc-tions of mercury and potassium iodide internally, increased rapidly to 30 grains three times a day. On October 11th (see Plate I, Fig. 2) the pain was referred to the left side of the chest more than to the right, and in conjunction with

Plate I.   Med. Chir. Soc. Trans. Vol. LXXXI
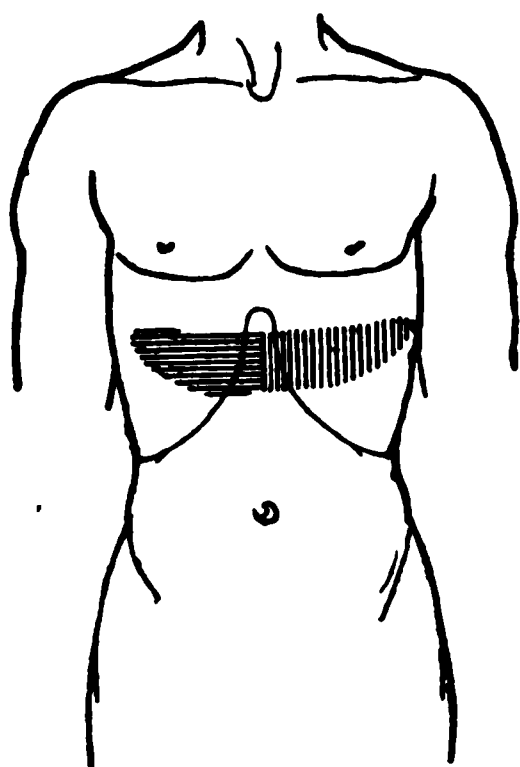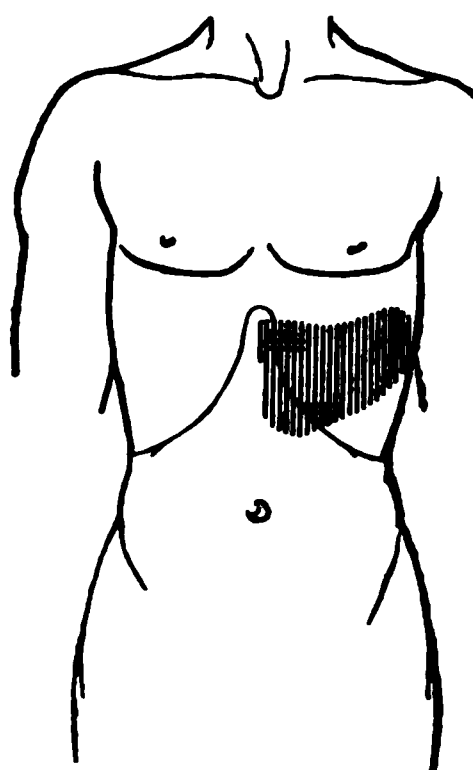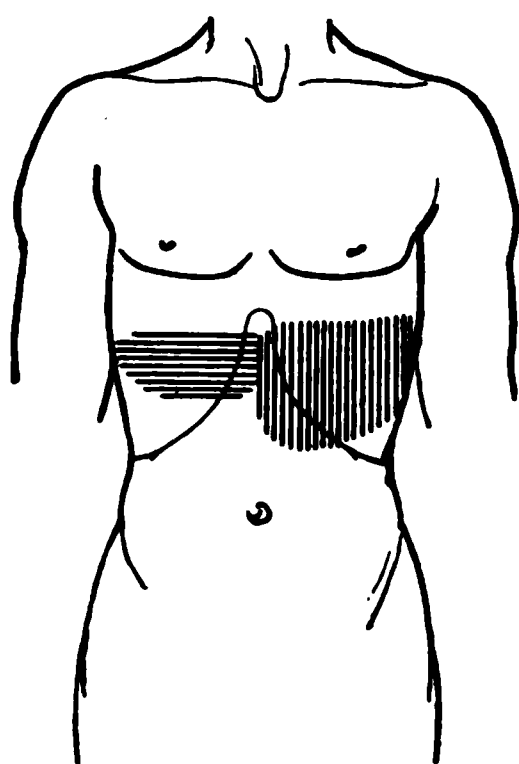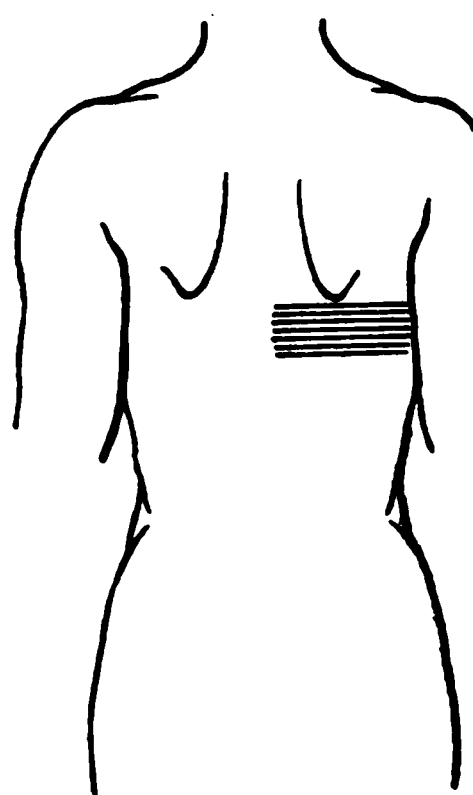
FIG. 1.   FIG. 2.

FIG. 3.   FIG. 4.

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Fig. 1.—Area of altered sensation as found on the patient's admission Sept. 14.

Fig. 2.—Condition on Oct. 11.

Fig. 3.—Condition on Oct. 18 ; anterior view.

Fig. 4.—Posterior view ; condition on Oct. 18.

The horizontal shading indicates hyperæsthesia.

The vertical represents partial anæsthesia.

this, hyperæsthesia of an extreme degree had developed over a triangular area corresponding to that originally noted on the right, but extending below the costal margin for about two inches. On the same day patient complained of difficulty in swallowing, referring the stoppage to the mid-sternal region. He stated that he had to drink some water after taking solid food before he could get it down. On October 12th the pain was referred alternately to the one side and the other. October 18th. —Plate I, Figs. 3 and 4 show the condition observed on that day. On October 21st patient complained that everything that he ate or drank burned him inside.

On October 23rd patient was being washed in bed, when he called out and instantaneously lost power in his legs. He described his sensations as being like a lightning shock all through him; the pain seems to have been intense. Immediately afterwards his left leg and thigh were completely paralysed; the right lower limb retained a trace of power, for he was able to move his toes and his knee slightly. When seen some hours later his condition was as follows :

He was quite unable to raise himself or even to turn over in bed. The abdominal muscles were acting, but the lower intercostals were completely paralysed; at all events, there was no movement of ribs from the sixth downwards, except that imparted by the diaphragm. The abdominal reflexes were brisk, the epigastric absent. Retention of urine existed for the first time; the bowels had not acted since the attack. In the lower limbs the condition was very much that described immediately after the onset of the paralysis, but some improvement had taken place. In the left, motor power was all but completely absent; the only sign of it lay in slight movements of the toes. On the right side a certain degree of power remained in all parts of the limb, but the movements were excessively feeble. As regards sensation, little if any alteration could be detected above the knee; below that level there was very considerable blunting of sensation of every kind without

complete abolition anywhere. This blunting was more marked on the left side than on the right. The condition of sensation over the zone of hyperæsthesia, to which frequent reference has been made, is depicted in the diagram, Plate I, fig. 3. Plantar reflexes could not be obtained on either side, but the knee-jerks were brisk. There was no clonus and no rigidity.

It was at once decided that laminectomy should be performed, but in view of the improvement that had taken place in the patient's condition during the day, and the great disadvantage of operating by artificial light, it was deemed wise to wait till the following morning.

Immediately before the operation on October 24th the condition remained unaltered.

October 24th (*Operation*).—The patient being placed in a convenient position and anæsthetised, the usual steps in the performance of a laminectomy operation were carried out (see Plate II). The laminæ of the sixth, seventh, eighth, and ninth dorsal vertebræ were removed. After a little dissection of fat and areolar tissue the theca was exposed, and it at once became evident that the upper part of the spinal column was free of and completely separated from the lower between the sixth and eighth vertebræ. The finger was passed by the side of the theca into the space which should have been occupied by the body of the seventh dorsal vertebra, but which now was filled by a mass of recent red currant jelly clot. This clot reached the spinal theca in front, and was supposed to have been due to sudden hæmorrhage from or into a sarcomatous tumour. The finger was passed more than once into the clot mass as far as possible, and no idea presented itself to the mind at the time that a search was being instituted into the interior of an aortic aneurysm. No hæmorrhage occurred. The wound was closed, and dressings applied in the ordinary manner. While the operation was proceeding it was noticed that after the removal of the laminæ the lower ribs, which had previously been paralysed, had resumed their function.

Plate II.   Med. Chir. Soc. Trans. Vol. LXXXII.
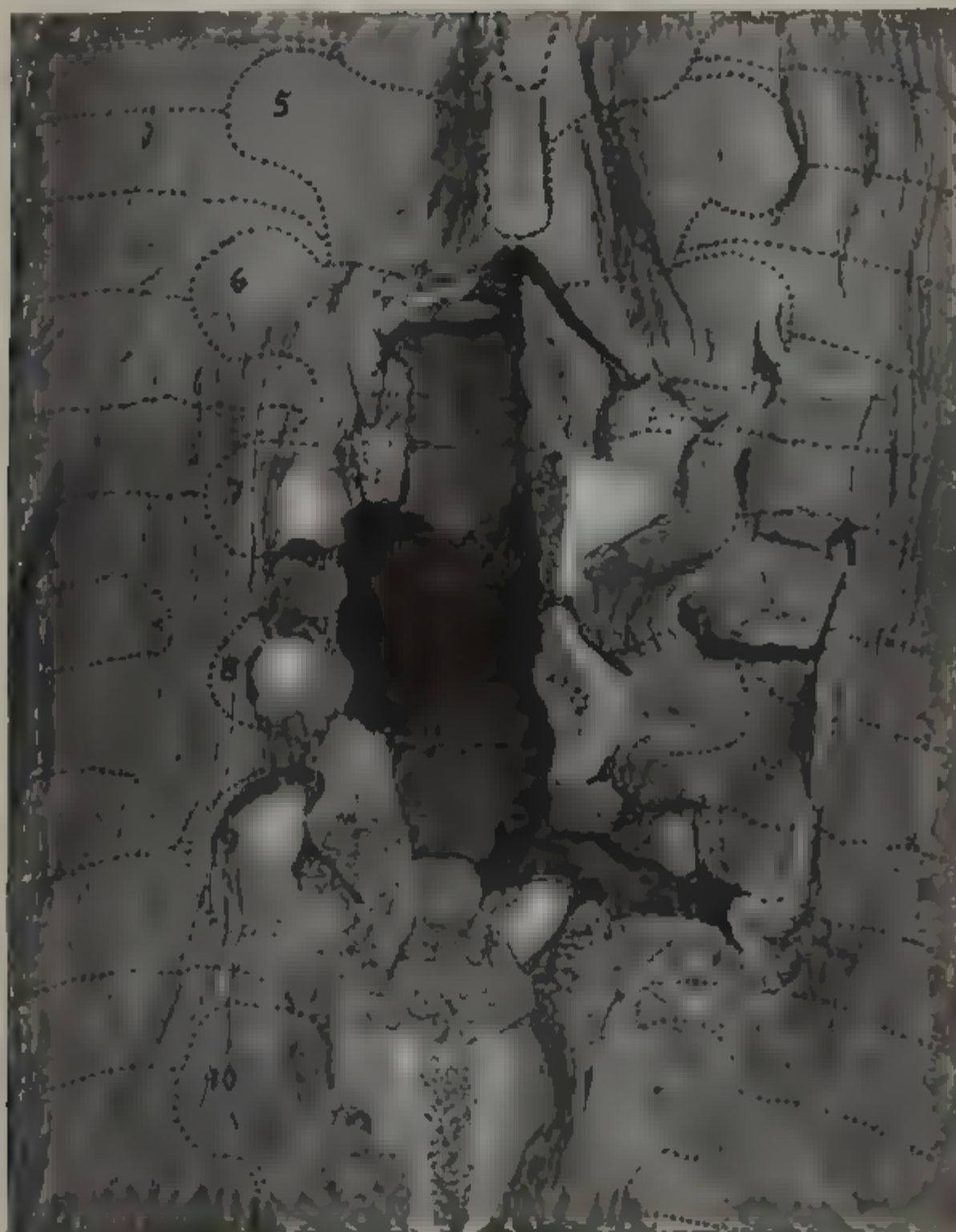
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(⅞ nat. size.)   The region of the operation is exposed.   The spinal theca was
removed before the drawing was made.   The opening of the Aneurism
into the Spinal Canal by the pressure destruction of the body
of a vertebra is clearly seen

M. H. Lapage, del.   Bale & Danielsson, imp.

After the operation the patient was taken back to th ward in a state of great collapse, from which he neve really recovered. Hypodermic injections of strychnin were given, and as much in the way of stimulants by th mouth as was possible, but in spite of this life was pro longed for eight or ten hours only, and death occurre without any return to full consciousness. Unfortunatel the patient's condition precluded any attempt at furthe examination.

Post-mortem examination.—The body is that of a poorl nourished man ; rigor mortis present.

Thorax.—The thorax being opened, the heart appear lying in the normal position, and overlapped by the lung as usual. The organ is of normal size and proportions and appears to be altogether free from disease. Th same may be said of the lungs and pleuræ.

On the *inferior* concavity of the transverse arch of th aorta is a small aneurysm. It is of the size of a sma sweet chestnut, which may be imagined to be stuck on t the artery by its flat surface. The small bag thus forme is completely filled by dense white laminated clot ; th aneurysm is, in fact, in the condition commonly mistake for one of cure. From the posterior aspect of the extrem lowest part of the descending arch another aneurysm ha sprung, but of a very different type. This forms tumour measuring vertically four and a half inches an transversely five inches. At the spot from whioh th aneurysm arises the aorta shows remarkably little sign c disease ; there is a hole cut in the posterior wall of th vessel, about an inch and a half in vertical measuremen and three quarters of an inch in transverse. Against thi externally the aneurysmal sac is, as it were, opposed. Th sac is full of clot (see Plates III and IV), which is mainl of the currant jelly type, though this is mixed with a goo deal of tough decolourised material. The wall of the sa has no definite layer of laminated fibrin in contact wit it. In position the tumour is exactly symmetrical, reacl ing about an inch and a half on either side of the vertebræ

bodies. Its upper border is very slightly above the aperture of communication with the vessel, and corresponds to the fifth dorsal vertebra, while the lower is as low as the tenth. Over its anterior surface runs the œsophagus, much stretched and flattened out.

Spinal column (anterior view).—The body of the sixth dorsal vertebra is eroded, that of the seventh is practically destroyed, and in places the finger comes down on the posterior ligament; even the intervertebral disc has disappeared. At this spot there is a complete solution of continuity in the spinal column. The destruction of the eighth dorsal vertebra is considerable but not complete, while the ninth is eroded like the sixth.

The ribs are affected on the left side from the sixth to the eleventh, while on the right side the change extends only from the sixth to the tenth. The heads of the seventh and eighth ribs and their bodies for an inch or more outside this are destroyed, while in the others the destructive process has not advanced beyond a more or less considerable erosion (see Plate III).

Spinal column (posterior view).—The laminæ have been removed from the sixth to the ninth dorsal inclusive. At the level of the seventh the finger can be passed through the anterior wall of the spinal canal straight into the cavity of the aneurysm; still there appears not to have been any considerable escape of clot or blood into the canal. At this level the upper part of the column moves freely on the lower in an antero-posterior direction, though the internal ligaments are not altogether destroyed. The upper portion of the spine is dislocated backwards, so that there is a sharp angle at this point.

The spinal membranes are healthy.

Spinal cord.—This does not appear flattened out, but at the spot (the level of the seventh dorsal) where it has to follow the sharp bend of the bony canal there is a section about half an inch in length, the tissue of which feels distinctly soft. [The cord was placed in Müller's fluid for further examination.]

Plate III.

Med. Chir. Soc. Trans. Vol. LXXXII
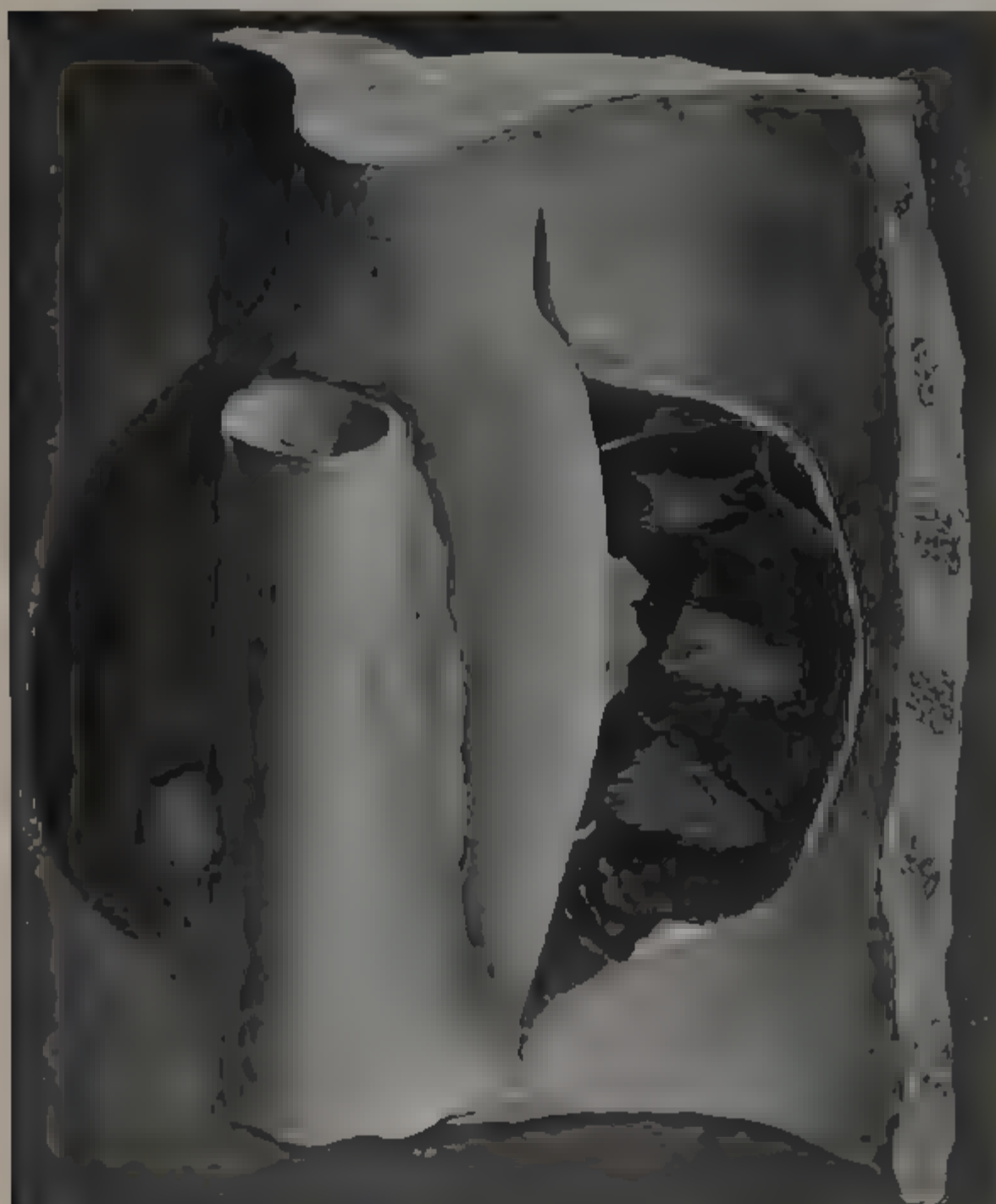
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(⅓ nat. size.) View of Aneurism and descending Aorta from the front
Through the window made on the wall of the Aneurism the Erosion
of the ribs is seen
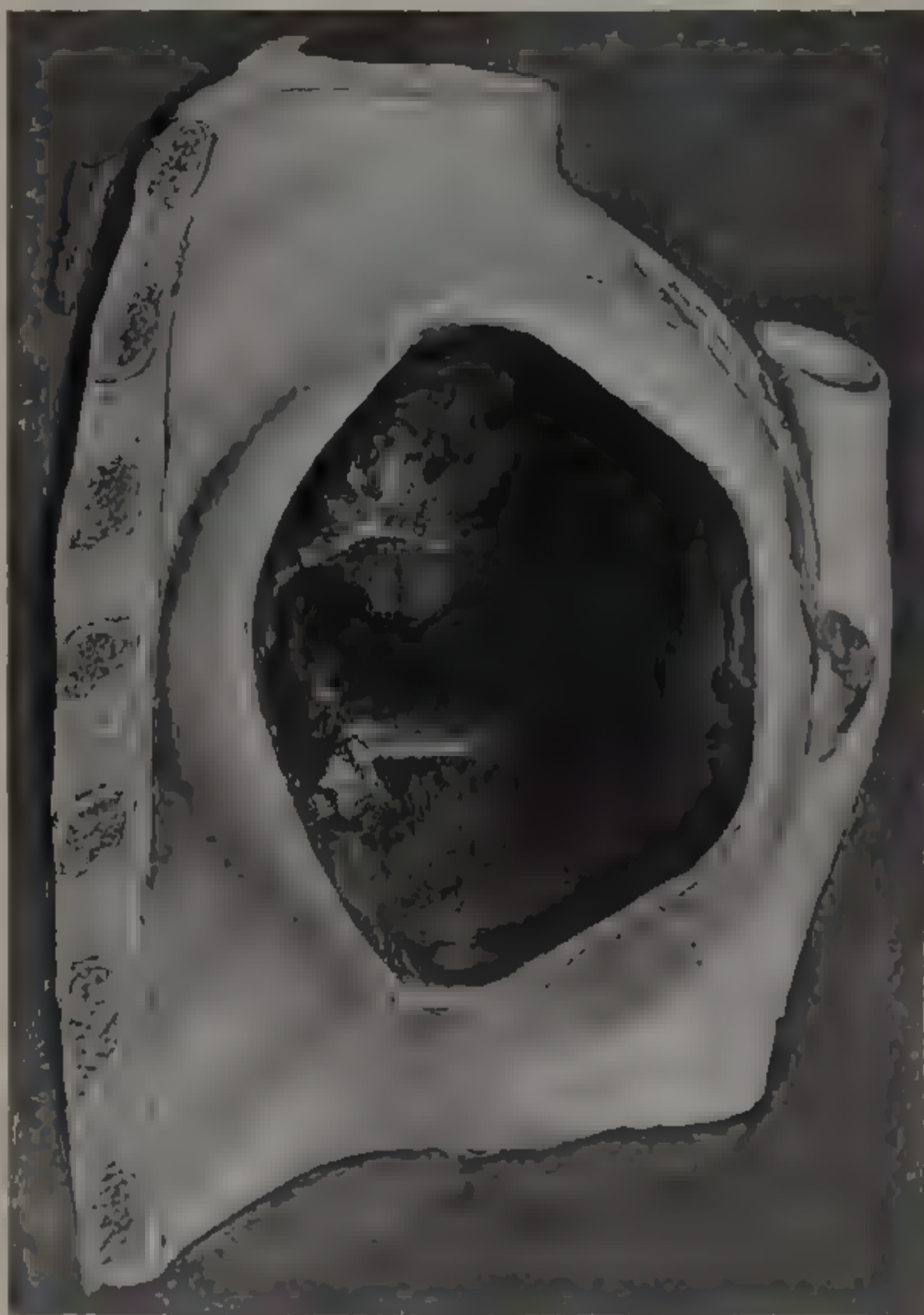
Dr. TIERNEY and Mr. BALLANCE's Paper

(⅓ nat. size.)   A portion of the wall of the Aneurism has been removed on the right side   The disappearance of the body of one vertebra and the erosion of others are shown.

Nerve-roots.—The only two to show any change visible to the naked eye are the seventh roots on either side. The left posterior root in particular is swollen and bright pink in colour. Its fellow on the opposite side is similarly altered but to a less extent, while the anterior root on that side (the right) shows as great change as the posterior root on the left. No change can be detected in the eighth roots on either side. The abdominal viscera are normal.

Microscopical examination (by Dr. PURVES STEWART).— Sections of the seventh left posterior dorsal root showed its blood-vessels to be markedly engorged, as compared with those of the normal fifth posterior root. There was no obvious connective-tissue proliferation between its nerve-fibres.

No degeneration was found in any of the nerve-roots nor in sections of the spinal cord, as high as the fifth and as low as the eighth dorsal segments, either by the method of Marchi or by that of Weigert-Pal.

Remarks.—It will have been gathered from the foregoing account that the case was one which throughout presented remarkable difficulties in the way of a correct diagnosis. The physical signs pointed strongly to some gross disease of the spinal column either primary or secondary. The symptoms, however, had remained absolutely stationary ever since their first appearance no less than two years before, and were limited in the most striking manner to those associated with irritation of posterior nerve-roots. No final conclusion was reached, but the diagnosis appeared to lie between pachymeningitis on the one hand and aneurysm, growth, and tubercular caries on the other. But whichever was accepted considerable difficulties had to be met. As against pachymeningitis the unusual and strict localisation of the lesion, and the failure of the hypothesis to explain the condition of the spine, had to be taken into account, while in favour of it was the history of previous syphilis. It

was finally put out of court by the negative results of a thorough course of specific treatment.

The possibility of caries was thoroughly discussed and rejected. Clinically the case did not correspond in the least with any example of that disease which either of us had seen or heard of. The completely negative result of the hot sponge test, which is by some regarded as all but pathognomonic, counted against it, while the persistence of symptoms of irritation without any signs either of advance or retrogression for so prolonged a period seemed practically conclusive.

A most careful examination failed to reveal any evidence, direct or indirect, of the existence of an aneurysm; the heart was normal in every respect, the sounds were not conveyed with any unusual force to the back, and the symptoms had from the first been symmetrical. Moreover the condition of the spinal column was hardly that which one would regard as compatible with aneurysm. Taking everything into consideration, the chances seemed to be in favour of growth. The length of the history and the unchanging character of the symptoms offered difficulties which were great indeed, but still less insurmountable than those which appeared to confront every other diagnosis. The great loss of flesh which had undoubtedly occurred was strongly confirmatory, and the transient difficulty in swallowing was at least as compatible with growth as with aneurysm. When the final attack of paraplegia occurred doubts became almost certainties, and even on the operation table nothing was found which appeared inconsistent with the theory of growth.

Little remains to be said on the neurological aspect of the case; our knowledge of the distribution of sensory areas has been made so complete by the researches of Sherrington and Head that no difficulty arose in deciding which nerves were involved. It is satisfactory to note that the localisation was confirmed by the autopsy. From the first it was plain that the seventh dorsal posterior root,

and later also the eighth, were the sufferers, and so it proved.

The sudden attack of paraplegia was in all probability the result of pressure by the dislocated vertebræ; the slight movement in bed was sufficient to rupture the last remaining bony link between the upper and lower sections of the spinal column. At the same time it is possible that the sudden access of symptoms was due to a rapid extension of the aneurysm.

It is noteworthy that at the time of the operation the finger passed within the sac without traversing anything of the nature of a limiting membrane, the clot alone preventing the outrush of blood. The destruction of the intervertebral discs by the pressure of an aneurysm is so rare an occurrence as to merit special notice. In caries, as is well known, the discs between the affected vertebræ disappear at an early stage of the disease; in aneurysm, on the other hand, the bones suffer, while the discs remain intact.

(For report of the discussion on this paper, see ' Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. xi, p. 43.)

GONOCOCCUS JOINT DISEASE IN INFANTS

SECONDARY TO PURULENT OPHTHALMIA

WITH TWENTY-THREE CASES

BY

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In the 'British Medical Journal' of February 28th, 1885, more fully on July 11th, 1885, and again, with the addition of a third case, in the same journal on October 10th of that year, I drew attention to a form of joint disease in infants which previous to that date had not been recognised or described.

The first case, an infant eighteen days old, suffering from purulent ophthalmia, was referred to me by a colleague on account of an acute arthritis of the left knee and a milder form of inflammation of the left wrist. It at once struck me that the very acute inflammation of the knee, indicated by great pain, redness, and swelling (resembling a joint in which suppuration was threatening,

or had already taken place), must have some definite rela-
tion to the conjunctival inflammation. The case reminded
me of some very acute joint affections I had seen in asso-
ciation with gonorrhœa among adults, one especially in a
woman whose knee-joint was pronounced by two very
eminent surgeons to contain pus, yet it subsided without
being opened; and another in a young man who was
placed in the isolation ward at Guy's Hospital, under my
care, as a case of erysipelas of the foot, yet in his case
also, when the gonorrhœa was discovered and treated,
the joint affection rapidly subsided without suppuration.

With these and similar cases in my mind, I felt con-
vinced that the joint affections of this newly-born infant
were dependent upon the purulent ophthalmia from which
he began to suffer two days after birth, and in my first
communication I wrote as follows :—" I am not aware
that any connection between ophthalmia neonatorum and
synovitis has ever been observed or described ; but there
seems no just reason if, as is generally supposed, the
synovitis of gonorrhœa is the result of absorption of
morbid products from the urethral mucous membrane, why
the conjunctival mucous membrane should not offer an
equally favorable absorbing surface. It is scarcely
probable that the inflammation of these two joints could
be referred to any other cause ; and in my own mind
there exists no doubt whatever that this is a case of
gonorrhœal rheumatism consequent upon absorption from
the conjunctival surface."

It will be remembered that Neisser first described the
gonococcus in 1879, but it was not till 1885, the year my
observations were published, that Bumm proved the
pathogenetic relation of the microbe to the discharge, so
that " absorption of morbid products " was the general
term I used for what would now signify the entrance of
the gonococci through the conjunctival mucous membrane
to the lymphatics or blood-vessels.

As to the inoculation of the gonococcus in my first
case there could be no shadow of doubt, for all three

patients—father, mother, and infant—who contracted the contagion in succession were under hospital treatment at the same time. I was very careful to exclude the possibility of the joint affection being caused by syphilis, and of this disease there was no evidence whatever. Treatment was directed towards the cure of the ophthalmia, and merely an evaporating lotion was applied to the knee, the inflammation of which had completely subsided at the end of five weeks.

Commenting on the difference in severity of the inflammation of the wrist compared with that of the knee, I threw out the suggestion that further experience would probably show that there were in infants two forms of joint disease caused by infection through the conjunctiva —a very acute arthritic variety and a mild form of synovitis causing effusion without redness, corresponding to the forms met with in gonorrhœal rheumatism of adults, and that the milder form in infants would be liable to be overlooked. "I make this suggestion," I wrote, "not merely from analogy, but because the inflammation of the wrist in my first case never reached the height of the inflammation of the knee, and therefore the two forms may possibly be found in the same individual, or the type may be throughout of the milder kind."

The third case, which I published in the 'British Medical Journal' of October 10th, 1885, proved the correctness of this prophecy. The infant, born of a mother who had suffered from a purulent vaginal discharge for two months before delivery, began to suffer from purulent ophthalmia two days after birth. A fortnight later its left knee was noticed swollen, and the child cried if the limb were moved or touched. When I saw it there was a good deal of effusion but no redness, and with the cure of the ophthalmia the synovitis subsided.

The study of these cases, therefore, showed that the ophthalmial rheumatism of infants may attack the joints in two distinct forms, viz. (1) as a very acute arthritis,

accompanied with much swelling, tenderness, and redness, strongly suggesting a tendency to suppuration ; and (2) as a subacute synovitis, giving rise to a good deal of effusion, and pain on movement, but to little or no surface redness.

On October 31st, 1885, Mr. R. G. Fendick, of Clifton, published in the ' British Medical Journal ' a fourth case in support of my views. His patient was the child of parents both suffering from gonorrhœa. The ophthalmia commenced on the third day after birth, and ran a rather severe course. During the third week after birth Mr. Fendick was sent for to see a swelling of the child's knee, and found the left knee full of fluid, semi-flexed, hot to the hand, with pain on movement. The father had not had syphilis, and three elder children were healthy and robust. The treatment adopted was purely local, and with the cure of the ophthalmia the inflammation of the knee rapidly subsided. From that time up to the present, a period of thirteen years, I believe no new case has been published by any British surgeon. The observation appears to have fallen quite dead in this country, and writers of text-books have, for the most part, either entirely ignored the original memoir or feared to commit themselves upon it. Mr. C. N. Macnamara, however, in the third edition of his work ' On Diseases of Bones and Joints,' published in 1887, quoted very fully my observations, but this stimulated no further inquiry ; and as about this time I ceased to attend out-patients, both at Guy's Hospital and the Evelina Hospital for Children, I lost the opportunity of adding to the number of my cases.

Happily, as not unfrequently happens, the newly described disease attracted more attention abroad, and I am now able to give an analysis of upwards of twenty cases, collected from various sources ; and the publishers of these reports almost invariably give me the credit of having made the first observation of the disease.

Shortly after my second communication to the ' British

Medical Journal,' Dr. Philpot published in the 'Lancet' of October 6th, 1885, the case of a child aged nine years, who had certain joints affected in association with vulvo-vaginitis of gonorrhœal origin. These cases occur in older children, and are more strictly analogous to the gonorrhœal rheumatism, of adults. Although in Dr. Philpot's case the vulvitis was traceable to defilement by a young man suffering from gonorrhœa, in other more numerous cases the vaginal inoculation appears to have been traced to sponges, towels, or diapers used by those suffering from gonorrhœa, or to secondary inoculation from purulent ophthalmia. These cases are not uncommon between the ages of two and ten years, and should be kept distinct from the ophthalmial rheumatism of new-born infants, which I wish more especially to draw attention to. Dr. Vignaudin, in the 'Revue Mensuelle des Maladies de l'enfance' of May, 1895, gives a very good account of these two classes in which Neisser's gonococcus plays the chief part.

ORIGIN OF THE OPHTHALMIA.

Of the twenty-three cases of ophthalmial joint disease, collected and briefly reported at the end of this communication, it will be found that eighteen were due to ophthalmia neonatorum caused by inoculation from mothers who were suffering from vaginal discharges at the time of labour. The remaining five had the conjunctivæ inoculated at a later period.

The date on which the purulent discharge from the eyes was first noticed was in two cases the second day after birth, in five cases the third day, in two cases the fourth day, whilst in one case it did not appear till the fifth day in one eye, and extended to the other eye on the seventh day after birth. In the other cases the exact date of the appearance of conjunctivitis is not noted. From these cases it would seem that the third day is that on which purulent ophthalmia most gene-

rally declares itself.   Of the five secondary inoculations, Widmark's case caught the ophthalmia from another child on the sixteenth day after birth ; Debierre's case from a younger sister, who was suffering from ophthalmia neonatorum, hence probably three or four years old, though not stated ; one of Darier's patients was two years, the other four years of age.   Morax's patient was seven years of age ; she had been inoculated by the finger of a girl aged four suffering from vulvo-vaginitis.

TIME AT WHICH THE JOINT DISEASE APPEARS.

A study of the cases collected brings out very clearly that the date after the appearance of the ophthalmia at which the joint disease may be expected to show itself is towards the end of the second week or during the third week.   Thus in no less than eight cases the joint affection was noticed first during the third week of the ophthalmia (Widmark, Fendick, Darier [2], Lindermann, Escherich, Haushalter, Griffon).   Seven cases were first noticed to have inflamed joints during the second week after the appearance of ophthalmia, viz. two on the tenth day (Morax, Berenstein), four on the twelfth day (Lucas [2], Debierre, Deutschmann), one on the fourteenth day (Sobotka).   Thus the second and third weeks account for two thirds of the cases.   Of the earlier cases, putting aside the case mentioned by Ashby and Wright, on the testimony of the friends that the joint affection was present on the day of birth, two cases (Zatvorniski's and Tyrrell's) appear to have shown joint inflammation on the fourth day of the ophthalmia.   Of the cases late in appearance Hoëck noted one in the fourth week and another in the fifth week, whilst Davies-Colley's case did not appear until the thirteenth week of the ophthalmia.   With regard to the case briefly mentioned by Ashby and Wright, where " erysipelas of the hand " was noticed on the evening of birth and stiffness followed, I presume the wrist was injured at birth and became secondarily

infected through the conjunctivæ. The only other explanation of so early au occurrence of the joint disease would be that the membranes had ruptured a day or two before delivery, and the vaginal infection thus allowed to reach the eyes. Ophthalmia at birth has been noticed by Parichew in a case where the membranes ruptured three days before birth (' Revue générale d'Ophth,' 1893).

THE JOINTS MOST LIABLE TO BE AFFECTED.

Although taken collectively the cases show that there are few joints which in some one or other case may not have escaped, the disease shows a very decided predilection in favour of the knees, which were inflamed in no less than fourteen cases. Moreover, there was a great preponderance in favour of the left knee, which was inflamed in ten cases. Next to the knee the left wrist has been noticed inflamed most often (six cases), but the elbows, ankles, hip, and joints of hand and foot have also been seen affected, and in two cases the tendon sheaths. The frequent occurrence of the disease in the left knee and left wrist cannot, I think, be due to chance, and requires some explanation. This I do not think we need go far to discover. It is known that sprained or injured joints have their resistance to the entrance of pathogenetic microbes lowered, and thirty per cent. of sprained joints in the lower animals become tubercular if the animal be injected with the tubercle bacillus in some other part of the body. Now mothers and nurses usually by preference carry the infant on the left arm so as to have their own right hand more free for use ; consequently whilst the infant's right arm and to a less extent its right knee are protected by the nurse's body, the left knee and left wrist of the infant are the joints most exposed to cold and injury. I believe this to be the explanation, the most exposed joints being those most liable to injury and to infective inflammation.

Course and Duration of the Inflammation.

The joint affection being secondary to a gonococcus infection of the conjunctival mucous membranes, the disease will have a tendency to subside as the germinating centres on the conjunctivæ become disinfected. Thus we find in Tyrrell's case, where the infant was born in hospital and received immediate and active treatment, the ophthalmia was cured in nine days, and the joint disease, which commenced on the fourth day of the ophthalmia, began to subside five days later. On the other hand, in cases where the ophthalmia is less early, less actively, or less effectively treated, the joint disease is likely to be slower in its tendency to resolution. The acuteness of the ophthalmia, however, does not appear to bear any relation to the occurrence of the joint disease; but it would rather seem that a lack of resistance on the part of the infant, or perhaps some slight injury, determines the joint inflammation. Though some few have recovered in from ten days to a fortnight, the majority run a course of from three to five weeks before complete resolution takes place.

Complete resolution may, however, be always looked forward to with confidence, even when the inflammation is so intense as to suggest suppuration in the joint. This complication very seldom occurs, and the resolution is so complete, that as a rule no loss of mobility follows. In the cases where more serious results occur I make the suggestion that the effects of another microbe have been added to those of the gonococcus.

Relation of the Gonococcus of Neisser to the Joint Affection.

Darier, in the year 1889, appears to have been the first to show the presence of the gonococcus in the secretion from the conjunctiva in these cases of ophthalmia neonatorum, but Deutschmann in 1890 went further, and by aspirating an inflamed knee of an

infant three weeks old suffering from purulent ophthalmia was able to show the presence of the gonococcus in the secretion of the joint. Thus the chain of evidence was completed. It may be taken for granted, then, as conclusively proved by the evidence of these and of other observers who have followed them, that the joint disease which I described in 1885 is, as I then indicated, dependent upon a gonoccocus infection of the conjunctivæ spreading therefrom and secondarily affecting the joints. In six cases in which joints were aspirated by various observers, the gonococcus was found in the secretion of the joint.

The Question of Double Infection.

It will be within the experience of all present that gonorrhœal rheumatism of adults may be seen to give rise to inflammations of joints of varying intensity, but that like the inflammations due to acute rheumatism (the microbe of which still remains undiscovered) the joints affected very rarely suppurate. Now, as I have shown, this is equally true of the ophthalmial joint disease of infants; but it will be noticed that three of the twenty-three cases collected ended in suppuration, viz. one of Hoëck's, Sobotka's and Griffon's cases. This leads to another question of great bacteriological interest, viz. when suppuration takes place, are we any longer dealing with a pure gonococcus infection of the joint? Judged by the fact that the gonococcus inoculated on a mucous membrane like the urethra or conjunctiva is there sufficient to excite suppuration, one would anticipate that filtered through the vessels or lymphatics it might be likely to cause suppuration in joints also; yet this is admittedly rare. The question I would put is whether where suppuration of a joint takes place, we are not then dealing with a double infection—gonococcus inflammation *plus* the inflammation of some other microbe ? Now it is very remarkable that in two out of the three cases in which suppuration occurred, another microbe

was actually identified in association with the gono-coccus. In Sobotka's case, which recovered, strepto-coccus was identified, and in Griffon's case, which died, staphylococci were found. These facts should lead to further and more exact bacteriological examination in the cases where suppuration occurs.

DIAGNOSIS.

The joint affections with which this might possibly be confounded are those due to syphilis, acute rheumatism, tubercle, and fevers.

Syphilitic joint disease, dependent on hereditary taint, is that with which the confusion would be most likely to be made; and, indeed, when I first described the disease several of my friends thought I was probably overlooking this possible cause of joint inflammation, and it was argued that as the mother was unfortunate enough to acquire gonorrhœa she might be suspected of having acquired syphilis also.

In almost all the cases of ophthalmial joint disease the inoculation of the conjunctiva takes place at birth, and the joint affection follows soon after, whereas syphilitic joint disease rarely appears till towards the end of the first year, and is then commonly associated with enlarge-ment of the epiphyses and pseudo-paralysis of Parrot. The gonococcus inflammation is more painful, and the syphilitic is preceded by other signs of syphilis. In Moncorvo's case there existed ophthalmia, vulvitis, and syphilis. Taken alone it would be scarcely worth includ-ing in this series, but as the ophthalmia appeared first and the vulvitis was a secondary inoculation I have included it, and the rapid course run by the joint inflammations points to gonococcus rather than to syphilis as the cause.

Acute rheumatism seldom occurs in children before the age of four or five. It is accompanied by acid perspira-tions and prolonged fever, and is influenced by the administration of salicylates. The ophthalmial rheuma-tism attacks few joints, occurs very early (unless due to

secondary infection), is uninfluenced by salicylates, and bears no relation to a rheumatic diathesis. According to M. le Dr. Vignaudin, in not one of the cases that he collected was there any history of rheumatism among the parents. It has a tendency to resolution as soon as the ophthalmia is effectively treated and completely cured.

From *tubercular disease* it is distinguished by its more rapid course, greater pain, more effusion and redness, and associated purulent ophthalmia. In any case, indeed, where an acute or subacute joint inflammation is found in association with purulent ophthalmia the probable dependence of one on the other should be taken into consideration.

From *joint affections following scarlet fever or other acute diseases* it is to be distinguished by the presence of the ophthalmia, the early age of the patient, and the absence of history of an exanthem. Only in cases of secondary inoculation, occurring in older children, would there be likely to be confusion.

The treatment should be directed mainly to the early and complete cure of the purulent ophthalmia, which is the infecting source. As soon as the frequent application of antiseptic lotions to the surface of the conjunctivæ has cured the ophthalmia, the joint disease may be expected to subside. Locally, evaporating lotions may be used to reduce the surface heat, or wool may be wrapped round to protect the joint from injury, and a bandage lightly applied. In young infants little can be done in the way of fixing the joint which in an older child would be of advantage. Now that the pathology of these cases has been clearly proved, I should deprecate the employment of an aspirator for mere curiosity.

Abstract of Twenty-three Published Cases.

No. I (Lucas, 'Brit. Med. Journ.,' Feb. 28th, 1885).—A male infant, aged 18 days, was brought to see me on February 12th, 1885, by his mother, M. B—, aged 34. She had been twice married. By her first marriage she had given birth to

five children, all of whom were living and healthy. None of these had suffered from rash or snuffles. She married a second time five years ago, and the infant brought was the second child of the second marriage. The elder of these was now 2 years and 6 months; she had suffered from no syphilitic symptoms. A week later she was brought for my inspection, and appeared perfectly healthy. The mother had never had miscarriage, rash, sore throat, or any symptom attributable to syphilitic infection.

About a fortnight or three weeks before her labour she noticed that she was suffering from a thick purulent discharge, and accused her husband of having infected her, which he admitted when she discovered he was attending at the hospital for a discharge from the urethra.

A day or two after birth the child was noticed to have a purulent discharge from the eyes, and was treated from the hospital with alum lotion. About a fortnight after birth, whilst the discharge from the conjunctivæ of the infant was still profuse, the mother noticed that its left knee was enlarged and painful, and that the child cried when it was moved. A little later the left hand was observed to drop, and the left wrist was noticed to be painful on movement.

On examination the knee was found greatly enlarged. It contained a considerable quantity of fluid, so that the patella floated, and was sufficiently red to indicate a possible tendency towards suppuration. The swelling was not merely a distension of the synovial membrane, but the enlargement was equally distributed above and below, so that it could not be traced to inflammation of either epiphysis. The mother said the knee had gradually increased in size since she first noticed it painful. The wrist was enlarged but not red; it creaked on movement, and this caused pain, so that the child cried. The only local treatment employed was the application of dilute lead lotion over the inflamed joints.

February 19th.—The wrist was more swollen and slightly red. The knee was less inflamed, but not diminished in size. The purulent ophthalmia was still profuse. A stronger alum lotion (8 grs. to an ounce of water) was now ordered to be dropped into the eyes every half-hour after bathing away the discharge.

26th.—The effect of the increased strength of the lotion on the eyes is very marked. The purulent discharge has almost entirely ceased. The wrist is much better, and only very

slightly swollen. The knee is also better, but still large and somewhat hot.

March 5th.—The eyes were now well, and the corneæ were quite clear. The wrist had recovered, and could be moved without pain. The knee was very much less inflamed and less swollen; the redness of the surface had disappeared.

25th.—The inflammation of the knee had quite subsided, and the joint could be moved without pain.

April 9th.—The child was again brought up for inspection He had greatly improved in health and strength. The joints were well. He showed no signs of inherited syphilis.

No. II (Lucas, 'Brit. Med. Journ.,' July 11th, 1885).—For the following case I am indebted to my colleague, Mr. Davies Colley, who kindly hunted it up in an old note-book after discussing with me the case previously related. It was headed in his note-book "Pyæmic abscess of knee-joint in a newly-born infant;" but the subsequent history stated that the joint recovered without suppuration.

L. T—, aged 3 months and 2 weeks, was first seen on May 2nd, 1874. The mother had suffered from discharge ever since her confinement (seventh child). The child had bad eyes four days after birth. She attended under Mr. Higgens for gonorrhœal ophthalmia. The left knee-joint had been affected about eight days. She had now the left knee-joint red and full of fluid

May 13th.—There was diarrhœa. The knee was less swollen

June 3rd.—She was better. The knee was nearly well; it had not suppurated. The only treatment mentioned is Aqua Calcis

No. III (Lucas, 'Brit. Med. Journ.,' Oct. 10th, 1885).—A woman aged 26, brought her infant to see me on July 16th 1885, suffering from ophthalmia neonatorum and a swollen knee

She gave the following history :—She was married four year ago last October. Her first child was born at the seventh month on the last day of the following July. It suffered neither from rash nor snuffles, and lived to the age of seven months when it died of whooping-cough. Between this and the next she had an early miscarriage about the second month. The second child was born on March 9th, 1883, at full time and healthy. It suffered neither from rash nor snuffles, and lived to be a year and six months, when it died of measles. The

patient was her third child, born June 20th, 1885. The eyes were clear at birth, but two days later they began to discharge. The mother was given a lotion which she thought too strong, so bathed the child's eyes only with warm water about every half-hour. A fortnight after birth the child's left knee became swollen and painful, and it cried when the knee was moved or touched. About the same time a red rash appeared over the child's buttocks, which the mother attributed to the use of soda in washing the child's diapers. The diapers are made of coarse towelling. The eruption is a bright red vesicular eczema, confined to the region irritated by the child's excretions. There are no coppery shining spots characteristic of syphilis. The hands, face, and mouth are free from eruption, and the child has no snuffling at the nose. The mother suffered from a yellow discharge from the vagina for about two months before the birth of the child. She had never suffered from any skin eruption or sore throat.

The house surgeon saw the case at the first attendance, and ordered a borax lotion and a grey powder.

July 23rd.—First seen by me. The eyes were still red and secreting pus, but better. The rash on the buttocks was bright red and vesicular. The left knee was semiflexed and the joint distended with fluid, but the skin was not red on the surface.

This case ran a mild course, and with the cure of the ophthalmia the synovitis rapidly subsided.

No. IV (R. G. Fendick, 'Brit. Med. Journ.,' Oct. 31st, 1885).— In this case the father had had a gleet for eighteen months, and the mother a discharge from which she had been suffering for several months before her confinement. She was attended by Mr. Fendick in her three former confinements, and the children are all healthy and robust. The father has never had syphilis. The ophthalmia commenced on the third day after birth and ran a rather severe course, but eventually was cured by the usual astringent treatment combined with scrupulous cleanliness.

During the third week after birth Mr. Fendick was sent for to see a swelling of the child's knee, and found on examination the left knee-joint full of fluid, semiflexed, hot to the hand, with pain on movement. The treatment adopted was purely local— warm fomentations, &c., and latterly the application of a lini-

ment of iodide of potassium and soap. "The knee is now rapidl;
improving; the pain has subsided, the swelling is much less
and the child is beginning to use the limb with freedom, and
will I think make a good recovery."

No. V (Zatvornitski, ' Wjestnik Ophtal.,' 1885).—In a newly
born infant ophthalmia appeared first in one eye, and, through
carelessness and want of attention, it extended into the other
eye, and lasted about six days. When the ophthalmia was mos'
acute it was noticed that the right elbow and wrist became
swollen and inflamed, and shortly after the left ankle. The
child's temperature was 40° C., and it was very prostrate. The
inflammation of the joints continued for some ten days, after
which it gradually subsided.

No. VI (Debierre, L., 'Revue générale d'Ophthal.,' 1885
p. 209).—The author alludes to Mr. Lucas's original observation
in the 'Brit. Med. Journal,' which is abstracted, and relates the
following case from the clinique of Dr. Ed. Meyer.

Amedée D—, infected March 28th by his little sister, who was
suffering from ophthalmia neonatorum. Very severe purulent
ophthalmia of both eyes, with chemosis of the conjunctivæ and
infiltration of the corneæ followed. It was treated with scarifi-
cation, followed by cauterisation with mitigated silver nitrate
stick and lotions of 1 in 5000 of mercuric chloride. Conjuncti-
vitis healed without leaving any trace.

On the twelfth day after contagion the child was suddenly
seized with acute inflammation of the left elbow, swelling, and
violent pain on movement. The arm was rendered immoveable
by dressings, and tincture of iodine applied. The inflammation
lasted about three weeks. No other joint was affected.

No. VII (Widmark, Dr. Johan, 'Jahrbuch für Kinderheilk,'
vol. xxix, 1886, p. 157).—A boy born April 22nd, 1885, was
seized on the sixteenth day of his life with purulent conjuncti-
vitis, possibly by infection from another child. The conjuncti-
vitis was not acute, but obstinate, for in spite of daily treatment
it lasted two months. Gonococci were found in the secretion.

On the sixteenth day of the disease the right knee and foot
began to swell, and the skin over them was red and tense. The
swelling was very tender. Wet applications were put over the
swollen parts, and the limb fixed by means of a bandage. On

the eighth day the swelling had begun to diminish, and in fourteen days it had disappeared.

No. VIII (Darier, 'Archives d'Ophtal.,' 1889, p. 175, Case 1).—
A child 4 years of age, with very severe purulent ophthalmia.

At the end of the third week the mother noticed that the child cried when the right hand was moved. The metacarpo-phalangeal joints, and particularly that of the index finger, were red, swollen, and very painful. The inflammation subsided in eight days, and twenty days after the onset the movements of the joints were quite free and without pain.

No. IX (Darier, 'Archives d'Ophthal.,' 1889, p. 175, Case 2).—
A child of 2 years with purulent ophthalmia, the pus from which was found to contain gonococci.

About the third or fourth week the mother noticed that the child could not put the right foot to the ground, and that it cried night and day. The ankle-joint was found to be red, swollen, and very painful. Three or four days later the right wrist was also affected. The child had temperature 103°, the local temperature of the right wrist being 103·5°. Fifteen days after this acute fever the child appeared very weak and anæmic; the foot was almost well, but the wrist still remained red and painful. Salicylates had no effect upon the condition, which was treated with compresses of cotton wool and mercurial inunctions. In three weeks the joints were quite cured.

No. X (Deutschmann, 'Arch. für Ophth.,' 1890, p. 107, Case 1).—A boy 3 weeks old, suffering from ophthalmia neonatorum. This was severe and complicated by diffuse keratitis.

Twelve days after the onset of the conjunctivitis the child had right otorrhœa, and at the same time swelling of the right wrist and left ankle. The child subsequently succumbed to a cerebral condition following the otitis. No account is given of any examination of the joints.

No. XI (Deutschmann ('Archiv. für Ophth.,' 1890, p. 107, Case 2).—An infant 3 weeks old affected with ophthalmia neonatorum, in the course of which it was seized with an inflammation of the left knee. Gonococci were found in the secretion from the eyes, but could not be found in the vaginal discharge of the mother. A small quantity of fluid was drawn off from

the knee, and also found to contain gonococci. No other micro-organism could be found.

No. XII (S. Lindemann, ' Beiträge zur Augenheilkunde, June 1892, No. V, p. 30).—Child born December 6th, 1891, was brought to the Polyclinic on December 11th suffering from double purulent ophthalmia. After about three weeks the eyes had so far recovered that treatment was discontinued. On January 1st, 1892, the left knee-joint became considerably swollen, greatly reddened, and hot to touch. The joint was semi-flexed, and pain was caused by attempts to straighten it The hollows on the sides of the patella were filled up, and the joint was painful to touch. Temperature remained normal whilst under observation. The secretion from the eye was examined and found to contain a few typical gonococci. On January 7th, 1892, the knee-joint was punctured and blood-stained fluid withdrawn. In the cells of the joint-secretion diplococci were recognised. The joint was again punctured on January 9th, and an attempt made to cultivate the gonococcus but with only partial success. The termination of the case is not reported.

No. XIII (Morax, ' Progrès Médicale,' Oct. 22nd, 1892).— A girl of 7 years, admitted for purulent ophthalmia of the left eye with lesions of the cornea. This patient had been inoculated from the finger of a child of four, who was shown to be suffering from vulvo-vaginitis. Typical gonococci were found in the purulent secretion.

Ten days after the onset the patient complained of pains in the larger joints, chiefly in the left knee. The skin was hot and there was slight fever. The next day the left knee was swollen with well-marked effusion, a condition which lasted more than a week. It was treated with rest and the joints enveloped in cotton wool. Ten days after the commencement of the inflammation the girl could walk.

No. XIV (Escherich, ' Jahrbuch für Kinderh.,' Oct., 1893).— A new-born male child, badly developed, was noticed three day after birth to have gonorrhœal ophthalmia in the right eye.

In the course of the third week the left knee was swollen On admission the left knee was very swollen and held in position of flexion. After a week's rest in a splint the effusion

in the joint was completely absorbed. Then there was found a moderate swelling of the condyles of the femur. At the end of fifteen days the arthritis and the ophthalmia were completely cured.

No. XV (Hoëck, 'Wiener klin. Woch.,' Oct. 12th, 1893, p. 736, Case 1).—M. S—, a female infant, born January 13th, 1893, came under observation seven days later for ophthalmia neonatorum. The ophthalmia was very severe, there being a thick discharge in which gonococci were found. In spite of repeated douches with permanganate lotion and swabbing with a solution of silver nitrate (2 per cent.) the inflammation persisted, and led to perforation of the cornea with prolapse of the iris.

A month after admission on February 25th there was an abrupt rise of temperature and great pain and tenderness in the joints of both lower extremities. The next day the left knee was very swollen and presented distinct fluctuation. Two days later, as the pyrexia was maintained, the joint was aspirated and about 2 c.c. of sero-purulent fluid drawn off. This was found to contain numerous pus cells, in the interior of which masses of gonococci could be seen. The cultivations upon serum-agar yielded positive results. A second aspiration gave the same results.

On March 18th the left hip was very swollen, and presented abnormal mobility. The head of the femur appeared to be lifted out of the articular cavity; the mobility became more and more striking. The leg assumed the attitude of extreme adduction, flexion, and external rotation. The distinct projection of the trochanter on the outer side led to the conclusion that the epiphysis was separated.

The child succumbed on April 15th to an attack of broncho-pneumonia. The lesions in the eye were very marked; the anterior chamber was destroyed; the lids were swollen and covered with a yellow crust; the left cornea was opaque, and in its upper half there was a linear scar. The right nasal fossa was filled with pus. The left leg was flexed at the knee and hip. In the left knee there was a little muco-purulent fluid, and the synovial membrane was slightly swollen, œdematous, and pale. The right knee was normal. In the left hip there was slight swelling, with intense injection of the synovial membrane. The acetabulum was partly filled up. The round ligament was de-

stroyed, and under the anterior superior spine a new cavity was formed.   There was congenital dislocation of the right hip also.

No. XVI (Hoëck, 'Wiener klin. Wochen.,' October 12th, 1893, p. 736, Case 2).—M. K—, born April 22nd, was admitted on April 30th, 1893, with severe ophthalmia, gonococci being found in the pus.   The child improved until May 20th, when the temperature rose to 102·5°.   A small swelling appeared in the right ankle, and on the following day there was distinct fluctuation behind the external malleolus.   This swelling disappeared after the joint had been fixed in plaster of Paris.   On the 24th the temperature again rose to 103·8°, and the left ankle became much swollen.   This swelling subsided and the temperature fell; but a week later there was again a slight rise in the temperature, and the right elbow and wrist became swollen.   In the meantime the ophthalmia had been cured.   By June 26th the swellings of the joints had subsided and the patient was discharged to attend as an out-patient.   The child was four weeks old before the arthritis developed, so umbilical infection was considered out of the question.   There were no traces of hereditary syphilis and no evidence of any other disease.   These were the only two cases Hoëck had seen in four years complicated with arthritis.

No. XVII (Sobotka, 'Prag. med. Woch.,' 1893, p. 582).—A boy 5 weeks old.   On the third day after birth the child suffered from purulent conjunctivitis.   Both eyelids were œdematous, the conjunctivæ injected and the corneæ ulcerated.   There was a thick discharge from both eyes.   A fortnight later the child presented a small circumscribed swelling on the ulnar side of the left wrist.   This was red and tender, and three days later showed distinct fluctuation.   The wrist-joint was swollen, so that its natural power was lost.   At the same time the right ankle was inflamed, and abscesses appeared on the outer malleolus and the radial side of the left wrist.   Two of the abscesses were opened.   On admission there was thick purulent discharge from both eyes.   On the left side the elbow-joint and wrist were inflamed; on the right the shoulder, elbow, and wrist were all swollen, red, and tender.   Besides the abscesses already mentioned in the left wrist there was a fluctuating swelling on the inner side of the left elbow, and a similar abscess on the ulnar

side of the right wrist. In the lower extremities both ankles and right knee were swollen, and an abscess appeared over the external malleolus of the left ankle. There was also a small abscess of the size of a walnut in the subcutaneous tissue under the upper margin of the ilium about the centre of the crest.

The right hip and knee were flexed and the upper extremities strongly adducted. Passive movements caused acute pain, but there were no definite signs of effusions into the joints. From the pus of one of these subcutaneous abscesses gonococci and streptococci were found, but pure cultures could not be obtained. After incision the abscesses healed up, and the swellings in the joints subsided about three months after the onset.

No. XVIII (Moncorvo, 'La Médecine Infantile,' July 15th, 1894).—A female infant, aged 3 months when first seen, began to suffer from inflammation of the left eye three days after birth, and this spread to the right eye shortly after, and was accompanied by swelling of the lids and a yellow discharge. After a few days, on undressing the child the mother noticed a yellow discharge from the vulva. Under antiseptics the discharge from both eyes and vagina had ceased and only a redness of the conjunctivæ remained. About three weeks before seeking further advice the mother noticed the child cry when moved, and found the elbows, wrists, knees, and ankles red and swollen. Some of the joints had already recovered, the right knee remaining the worst. The child also presented the signs of hereditary syphilis. The child was treated with iodide of potassium and quinine. The inflammation of the joints subsided in the course of a fortnight. The mother admitted having a vaginal discharge before her delivery.

No. XIX (Haushalter, ' Revue Mens. des Mal. de l'enfance,' October, 1895).—A girl, 28 days old was admitted on June 1st, 1895. The child had suffered from purulent ophthalmia three days after birth. This was very severe and led to total blindness. Three and a half weeks after birth the mother noticed a swelling of the left wrist, and two days later there was a swelling of the right knee. On admission there was a marked swelling of the left wrist involving the dorsum of the hand. On the same side there was some swelling of the front of the forearm, apparently due to an inflammation of the tendon.

sheaths. On flexing the wrist on the forearm there was distinct sensation of creaking in the joint. The movement caused acute pain. The right knee formed a uniform round swelling. There was great distension of the synovial membrane by fluid, and distinct fluctuation. The fluid aspirated from the joint was not purulent, but contained numerous polynuclear leucocytes. A week later the swelling of the left wrist had subsided, the right knee was very much swollen, but fluctuation was still obvious. Purulent discharge from the eyes was much less. At the end of three weeks there was hardly any fluid in the right knee, but there was some enlargement apparently due to swelling in the bony extremities. The child was then discharged as cured. From the fluid obtained from the right knee gonococci were cultivated in beef broth, but re-cultivation upon the ordinary media was found impossible. Inoculation of the cultures in the bouillon into the peritoneal cavity of a guinea-pig had no effect.

No. XX (Griffon, 'Presse Med.,' No. 15, 1896).—A child three weeks old was admitted for purulent ophthalmia which was noticed a few days after birth. On admission the child had both double purulent ophthalmia and vulvo-vaginitis. The day after admission the wrist became swollen. The right wrist was flexed upon the forearm, the whole region was swollen, without any alteration in the colour of the skin, or elevation of local temperature. The dorsum of the hand was œdematous, subcutaneous veins being enlarged. Any movement of the joint caused acute pain. The right hip was flexed upon the abdomen, and could not be extended without great pain to the patient. The leg was flexed upon the thigh.

On examining the hip a distinct fluctuation could be felt behind the great trochanter, and the synovial membrane of the joint appeared to be distended with fluid. After a short rest this effusion quickly disappeared. The œdema and swelling of the right wrist subsided when the joint was fixed on a wooden splint. A week after admission the child's temperature rose to 103° F., and persistent vomiting and diarrhœa set in. Two days later the wrist, though not so swollen, was more deformed. The hand lay useless, slightly flexed upon the forearm. The flexor and extensor sheaths were both distended. There was a fluctuating swelling of the forearm near the styloid process of the

ulna. The hips remained flexed and adduction was limited.
Temperature at this stage was subnormal. Twelve days after
admission the child died. At the post-mortem examination a
thick, creamy, viscid fluid was obtained from the fluctuating
swelling on the forearm. The synovial membrane of the right
wrist, which appeared to be in communication with the swelling,
was very little altered, perhaps a little thick. The lesions were
chiefly peri-articular, the articulation being comparatively free.
In the right hip the lesions were mainly articular. The capsule
was found to contain a large quantity of very thick pus, the
peri-articular tissues appearing healthy. The articular carti-
lages had ulcerated in places, but in the main had preserved
their smooth aspect. The synovial membrane itself was smooth,
and not injected. The round ligament was destroyed. There
was no appearance of the capsule having been ruptured. The
other joints were healthy. The bacteriological examination
during life showed gonococci in the pus from the eye and
vagina, but pus aspirated from the joint yielded nothing but
staphylococci. At the autopsy the pus from the wrist and hip
was examined and cultivated, and undoubted gonococci were
found on the microscopic examination of the pus from these
joints, but only staphylococci could be cultivated therefrom.
No gonococci were found in the blood microscopically, and the
cultures from the blood remained sterile.

No. XXI (E. M. Tyrrell, 'Med. News,' March 7th, 1896,
p. 271).—A coloured woman suffering from discharge and with
a well-defined history of gonorrhœa, gave birth in hospital to a
well-developed female child. The child's eyes were carefully
cleansed and preventive treatment constantly applied. Four
days after birth slight puffiness and redness of the lids of one
eye commenced, and by the evening, in spite of hourly treatment,
the lids of both eyes were enormously distended, and slight
pressure caused pus to spurt from under them.

Four days after the appearance of the ophthalmia the wrists
and dorsum of both hands were slightly swollen, the left being
the worse. They were bathed in a hot saline solution, but the
swelling continued until it reached from the finger tips to the
elbows, and the skin of the wrists and forearms was œdematous.

The child appeared uncomfortable without indications of severe pain, except for a few minutes at a time.

Applications of a 10 per cent. preparation of ichthyol with belladonna were applied, and the hands and arms were bandaged.

Five days later the swelling had disappeared; the eyes in the meantime had yielded to treatment. At the end of three weeks the patient was discharged in good condition, though the ophthalmologist continued the treatment at the patient's home.

No. XXII (Ashby and Wright, 'Diseases of Children,' 1896).—"We have seen an infant a few weeks old in which a stiff flexed wrist remained as the result of what was described as "erysipelas of the hand." The swelling of the hand was noticed on the evening of the day the child was born, and it had also purulent ophthalmia."

No. XXIII (Berenstein, 'Centralb. für p. Augenheilk,' March, 1897, p. 84).—The case was that of the first child of an unmarried girl of nineteen years suffering from a vaginal discharge. The child, born at full time and strongly developed, was until the fifth day perfectly healthy, but on that day the right, and on the seventh day the left eye were seized with typical ophthalmia neonatorum. In the freely secreted pus numbers of Neisser's cocci were demonstrated. On the fifth day of the disease (i.e. the tenth of the child's life) the mother noticed a swelling of the right wrist, and two days later the knee of the same side was seized. The general condition was febrile. The joints were red and felt hot; handling of them made the child cry loudly. The knee was held flexed. Berenstein could not feel fluctuation. An exploratory puncture was "unfortunately" not allowed by the mother. The heart-sounds were clear. The ophthalmia recovered by the end of the tenth week of the disease without complications in either cornea or iris. The swellings of the joints lasted somewhat longer, but disappeared with equal completeness. The treatment used was painting with iodine and warm baths.

Note.—I have to thank my dresser, Mr. Stoëhr, for assisting me in obtaining several of these references.

Table of Cases, showing time of onset, joints affected, duration of the attack, and result.

Observer.	Age.	Onset of ophthalmia.	Onset of joint disease.	Joints affected.	Date of subsidence.	Result.
Lucas	18 days	2nd day	12th day	Left knee severely, wrist slightly	6 weeks	Complete recovery.
Davies-Colley	14 weeks	4th day	13th week	Left knee	5 weeks	,,
Lucas	1 month	2nd day	12th day	,,	1 month	,,
Fendick	3 weeks	3rd day	3rd week	,,	Not stated	Recovering when reported.
Debierre	Not stated	Inoculated from younger sister	12th day after inoculation	Left elbow and wrist	3 weeks	Recovery.
Widmark	32 days	16 days after birth inoculated from another child	16th day of disease	Right knee and foot	14 days	,,
Zatvornitski	Newly born	Shortly after birth	4th day	Right elbow and wrist and left ankle	10 days	,,
Darier	4 years	—	3rd week of ophthalmia	Metacarpo-phalangeal joints of right hand	20 days	,,
,,	2 years	—	3rd or 4th week	Right ankle and right wrist	8 weeks	,,
Deutschmann	3 weeks	Not stated	12 days after onset	Right wrist and left ankle; also right otorrhœa	—	Died from cerebral condition following otitis.
,,	3 weeks	Not stated	—	Left knee	—	—

Lindemann	3 weeks	Not stated	26 days after birth	,,	—	—
Morax	7 years	Inoculated from child of four	10 days after onset	,,	10 days	Could then walk.
Escherich	3 weeks	3rd day	3rd week	,,	15 days	Complete recovery.
Hoëck	7 days	Not stated; treated on 7th day; right cornea sloughed	5th week after birth	Left knee, left hip; knee aspirated twice; both joints suppurated	—	Died of broncho-pneumonia after 3 months.
,,	8 days	Not stated	1 month after birth	Right ankle, left ankle, right elbow and wrist	5 weeks	Recovery.
Sobotka	5 weeks	3rd day	14 days	Left wrist, right ankle, right shoulder, elbow, and wrist	3 months	Recovery after suppuration; gonococci and streptococci found.
Moncorvo	3 months	3rd day; a few days later vulvitis; also hereditary syphilis	2 months	Elbows, wrists, knees, and ankles	14 days	Recovery.
Haushalter	28 days	3rd day	3½ weeks after birth	Left wrist, right knee	3 weeks	,,
Griffon	3 weeks	Few days after birth; also vulvo-vaginitis	3rd week	Right wrist, right hip; also tendon sheaths	—	Died 12 days after onset; suppuration; gonococci and staphylococci found.
Tyrrell	Born in hospital	4th day	8th day after birth	Wrists	5 days; discharged in 3 weeks	Recovery.
Ashby and Wright	Few weeks	Not stated	Evening of day child was born	Wrist; "erysipelas of hand"	—	Stiff and flexed when seen.
Berenstein	Newly born	5th and 7th days	10th day after birth	Right wrist and right knee	5 weeks	Recovery.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. xi, p. 47.)

ON THE EFFECTS PRODUCED IN THE HUMAN SUBJECT

BY THE

ADMINISTRATION OF DEFINITE MIXTURE: OF NITROUS OXIDE AND AIR

AND OF

NITROUS OXIDE AND OXYGEN

BY

FREDERIC W. HEWITT, M.A., M.D.Cantab.

ANÆSTHETIST AND LECTURER ON ANÆSTHETICS AT THE LONDON HOSPITAL
ANÆSTHETIST AT THE DENTAL HOSPITAL OF LONDON

I. Introduction.

The effects produced upon the human organism by the inhalation of nitrous oxide gas, either pure or mixed with certain percentages of air or of oxygen, are of considerable interest; for not only do they afford those of us who employ anæsthetics for surgical purposes much food for reflection and study, but by reason of the sidelights which they throw upon the subject of asphyxia they deserve attention from physiologists.

Whilst most observers who have had much clinical

experience in the administration of pure nitrous oxide agree in the main as to the phenomena produced, very divergent views have been and are still expressed as to the effects which attend the inhalation of this gas with different percentages of air or oxygen. It was this fact, indeed, which led me some years ago to commence the investigation which I now have the honour of placing before this Society. It seemed to me that there was need for greater accuracy; that our knowledge should be of such an order that it would enable us to state that when nitrous oxide is administered with certain small percentages of air or oxygen certain phenomena will arise; that when these percentages are increased certain other symptoms will make their appearance; that so far as the asphyxial accompaniments of an administration of nitrous oxide are concerned these will fail to assert themselves when this or that percentage of air or oxygen is present; and that so far as anæsthesia is concerned this will be most satisfactory with certain mixtures, and unsatisfactory or unattainable with others.

It is not difficult to understand the wide discrepancies of opinion which have existed concerning the influence which different percentages of air and of oxygen have in modifying the effects of pure nitrous oxide. In the first place, errors as to the precise compositions of the gaseous mixtures employed are very liable to arise, either because the nitrous oxide used is not pure or because the gasometer with which the administrations are conducted is incapable of accurately measuring the proportions of the constituent gases. In the next place, it is not an easy matter to wholly prevent the ingress of the surrounding atmospheric air during the administration. This is probably the most fruitful source of error. I do not exaggerate when I say that years of practice are necessary before absolute accuracy in adjusting the face-piece or mask can be obtained. Persons with hair about the lower part of the face are, for obvious reasons, unsuitable subjects in an investigation of this kind; and

in the case of lower animals it is perfectly clear that if the gases be administered by means of a face-piece or mask, fallacies must inevitably result. It must also be borne in mind that the valves of the apparatus employed should work in such a way that no air can be sucked back during their closure, otherwise dilution of the mixture employed will occur. Many of the inhalers formerly used, and even several of those employed at the present day for administering nitrous oxide gas, are quite unsuitable for accurate administrations. In the third place, care must be taken that the channels' through which the patient inspires and expires are of sufficiently wide calibre, otherwise an asphyxial factor will be at once introduced, and the result thereby modified. In the fourth place, it is necessary, if we wish to obtain reliable and comparable results, to reproduce in each administration the main general circumstances which were present in other cases. Although it is true that we cannot, except rarely, obtain the same patient, we can, by employing the identical inhaler, method of administration, posture, looseness of dress, and general surroundings, secure such uniformity in our results that we are justified in making comparisons between the different percentages. From these considerations it will, I think, be clear that the possible sources of fallacy in administering gases and gaseous mixtures are very numerous; that if we wish to avoid them a close attention to numerous details is absolutely essential; and that unless they are avoided no reliance whatever can be placed upon the results obtained.

The present investigation was begun on December 3rd, 1894, at the Dental Hospital of London, Leicester Square, and was finished on January 26th, 1897.

II. The Gases and Gaseous Mixtures used in the Investigation.

There were in all 231 administrations, which I have arranged in two Series.   Series I consists of 226 cases; Series II of 5 cases.

The cases of Series I are comparable to one another, *i. e.* the general conditions in each case were the same, although the gases inhaled differed.

In Series II the cases were not comparable, *i. e.* the conditions under which the cases were conducted were dissimilar.

The following is a statement of the various gases administered in Series I and II.

Series I.

				Cases.
Pure nitrous oxide	.	.	.	20
,,	with	3 per cent. air	.	5
,,	,,	5 ,,	.	10
,,	,,	6 ,,	.	6
,,	,,	7 ,,	.	6
,,	,,	10 ,,	.	10
,,	,,	12 ,,	.	5
,,	,,	14 ,,	.	4
,,	,,	15 ,,	.	9
,,	,,	16 ,,	.	5
,,	,,	18 ,,	.	12
,,	,,	20 ,,	.	7
,,	,,	22 ,,	.	14
,,	,,	25 ,,	.	8
,,	,,	30 ,,	.	4
,,	,,	$33\frac{1}{3}$,,	.	1
,,	,,	3 ,, oxygen	5
,,	,,	4 ,,	.	10
,,	,,	5 ,,	.	17

Cases.

Pure nitrous oxide with 6 per cent. oxygen   11

,,   ,,   7   ,,   . 11

,,   ,,   8   ,,   . 18

,,   ,,   9   ,,   .   5

,,   ,, 10   ,,   . 10

,,   ,, 11   ,,   .   7

,,   ,, 13   ,,   .   2

,,   ,, 20   ,,   .   4

—————

226

Series II.

Pure nitrous oxide   .   .   .   2

,,   with 20 per cent. air   .   1

,,   ,,   5   ,, oxygen 1

,,   ,, 10   ,,   ,,   .   1

——

5

III. The Apparatus employed.

The gasometer which I used is here figured.

Its bell had a capacity of about 20 gallons, but, allowing for the " seal " (*i. e.* the layer of water necessary to prevent the escape of the gases when the bell was full), the working capacity proved to be exactly 18 gallons. The bell, which was 12 inches in diameter, was graduated by means of a painted scale into 100 equal divisions, so that any percentage of air or oxygen could be mixed with nitrous oxide. As the workable capacity of the bell was 18 gallons, each division of the scale represented ·18 gallon, and it was a simple matter to read off the scale to a tenth of each of these divisions. There were no counterbalancing weights, so that the mixed gases issued at slight pressure. The fulness of the inhaling bag, however, could be regulated at will by the tap between this bag and the gasometer. The
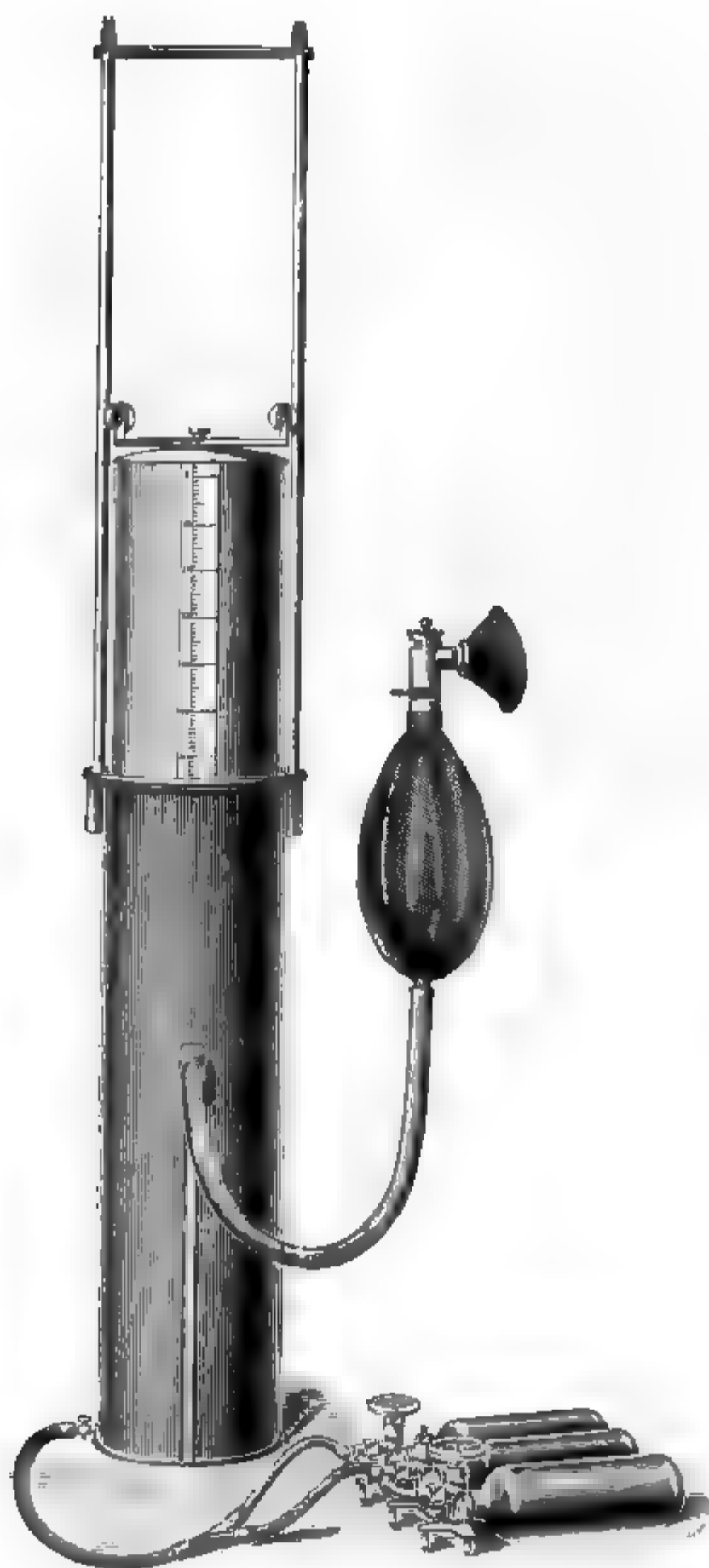
special stopcock through which the gases passed on thei
way from the inhaling bag to the face-piece was on
which I have elsewhere described. It contains tw
accurately working flap valves, so that the patient'
expirations escape into the surrounding air.

Before being used for any administration the bell o
the gasometer was always "washed out" by a stream o
pure nitrous oxide, in order to free it from all air. I
was then filled to about one half with the anæsthetic gas
the proper percentage of air or oxygen was nex
introduced; and finally the bell was filled up by mean
of more nitrous oxide. This system was adopted i
order to ensure the gases being thoroughly mixed
After waiting a minute or two the exit tube and inhalin
bag were "washed out" by allowing some of th
mixture to escape. The bag was then emptied, al
stopcocks were turned off, and the first reading on th
scale was taken.

IV. THE METHOD OF ADMINISTRATION; THE SYSTEM O
    NOTE-TAKING; AND THE TERMS USED IN THE COURSE O
    THE INVESTIGATION.

In all the cases of Series I the patients were place
in exactly the same posture, all constricting clothing wa
unfastened, the same directions were given as to breath
ing, the same questions asked afterwards, and the sam
apparatus used. The face-piece having been applied wit
accuracy, each patient was allowed to take two or thre
breaths of air through the stopcock. Instructions wer
always given to "take long, deep breaths backward
and forwards through the mouth." When it was foun
that the face-piece fitted well, the mixture was turned o
The bag was kept as far as possible half distended. Th
was done by regulating the flow of the mixed gases fro
the gasometer by means of the tap of the exit tub
Each administration was conducted till distinct signs c

anæsthesia occurred. The face-piece was then removed and the operation begun. The note-taking was continued till anæsthesia was at an end. The patient then left the chair, and the note-taker made a further entry as to the patient's sensations and opinions. The final reading of the gasometer was now taken, after pressing back into the bell any of the mixture left in the bag. Any after effects were noted.

The special circumstances of the cases of Series II are referred to in a later part of the paper.

Throughout the investigation I had the services of two assistants; one to count aloud the bells of a metronome, the other to enter, on previously ruled forms, the various observations which I made during the progress of each administration. The metronome was set to ring its bell every three seconds, and as one assistant consecutively called out the number of bells the other entered the notes at the various stages of the administration or resulting anæsthesia.

In taking notes of the cases particular attention was paid to the following symptoms :

1. *Anoxæmic convulsion.*—Various terms, such as " epileptiform muscular twitching," " clonic muscular spasm," " jactitation," and " oscillation," have been used by authors to denote the peculiar phenomena so common when nitrous oxide is administered free from oxygen. But as everything in this inquiry goes to prove that these muscular phenomena are due to absence of oxygen, I have used the term anoxæmic convulsion as the most appropriate by which to designate the condition. Convulsive movements occurring during the operation have been disregarded in analysing the cases, as such movements may arise from intercurrent mechanical causes.

2. *Duskiness ; lividity ; cyanosis.*—Alterations in the patient's normal colour are exceedingly common, not only under nitrous oxide itself, but under various mixtures of this gas with air or oxygen. In estimating the alteration in colour under nitrous oxide and its mixtures, I have

disregarded lividity arising during the operation period, for during this period it may, like anoxæmic convulsion, depend upon other influences than want of oxygen in the gaseous mixtures used.

3. *Stertor.*—All grades of stertor, from mild and barely audible snoring, to coarse, loud, irregular, guttural sounds, were met with and recorded. As in the case of convulsion and cyanosis, I have disregarded stertor during operation, as this may possibly be dependent upon causes other than those under consideration.

4. *Retching movements.*

5. *Phonation.*—Under this term I have included all expiratory vocal sounds, from the slightest to the most intense (shrieking). In order to place the cases as far as possible upon the same footing, I only took cognizance of phonation during the inhalation, and during the thirty seconds immediately following it.

6. *Excitement and reflex movements.*—Under this heading I have included all muscular phenomena other than the purely voluntary and the epileptiform above referred to. Thus, we may meet with uncontrollable nervous movements; intoxication movements; tonic spasm; fine tremor; peculiar movements of the arms, legs, or body, occurring late in the administration of nitrous oxide and oxygen ("secondary rigidity"); and those strictly reflex movements dependent upon the operation. I have only taken into account the occurrence of such muscular phenomena during the inhalation and first thirty seconds after. In a few cases forcible restraint was needed to keep the patient in the chair.

7. *After effects.*—I was unfortunately only able to ascertain the immediate after effects in the large majority of cases, as most of the patients did not return to the hospital.

8. *Laboured or " catching " respiration.*

9. *Unpleasant dreams.*

10. *Duration of anæsthesia after inhalation.*—It is an exceedingly difficult matter to say in any given case at

what precise moment anæsthesia comes to an end after an inhalation of nitrous oxide, pure or diluted. This is more particularly so when the operation has been completed in the first few seconds after the removal of the inhaler. It therefore follows that the number of seconds given as the duration of the available anæsthesia in each case can only be regarded as approximately accurate. In estimating the duration of the resulting anæsthesia I found the best plan was to repeatedly ask each patient, whilst recovering from the effects of the anæsthetic mixture used, to open her eyes, and to take the moment at which conscious response to this request occurred as indicating the cessation of anæsthesia. I may say that by adopting this plan one is usually certain of being well within the mark, so far as the perception of pain is concerned; for, as a general rule, there is, during the recovery from nitrous oxide anæsthesia, a distinctly analgesic borderland through which the patient passes after conscious response to questions has become possible (see p. 176). In several of the cases, either the patient's symptoms misled me, or, with the object of doing as much for the patient as possible, the operation was prolonged rather beyond the proper limit, and slight pain was felt. Deductions have, however, been made in these cases on the following system, it being held that the ideal to be aimed at is absolute unconsciousness, not merely of pain, but of any operative procedure whatsoever. Hence, whenever it was clear from the patient's statements that some pain was felt towards the end of the operation, or that consciousness was present without any pain being experienced, deductions were made for the erroneously estimated anæsthesia period. Two or three seconds were deducted when patients stated that they felt "something," but were unable to say precisely what they felt; or when they knew something of the end of the operation without experiencing any pain; or when they "thought" they felt something of the operation, but were not sure. See Cases 148, 62 a, 40, 122, 168,

146, 130, 133, 182, 121, 52, 18, 19, 97. Five seconds were deducted when slight pain was felt at the end of the operation, as in Cases 124, 33, 202, 95, 96, 206, 193, 5, 7, 22, 162, 103, 134, 42. Six or seven seconds were deducted when distinct pain was felt just at the end of the operation, as in Cases 84, 183, 8, 149, 76, 167, 99, 85, 91, 75, 78, 80. Ten, eleven, or twelve seconds were deducted when, from the patient's statements, and from a consideration of the operation performed, it seemed clear that an error to this extent had been made in estimating the anæsthesia. See Cases 156, 164, 77, and 48. In one case (Case 59) seven teeth were removed, but the patient declared that pain was experienced during the removal of the last three, so that the supposed anæsthesia of sixty seconds had to be reduced to thirty-six seconds. The following case will give a good example of the way in which the notes were recorded.

No. 169.	*Date.*—June 23rd, 1896.	*Place.*—Dental Hospital.

Weather.—Sultry morning. No direct sunlight.

Sex.—F.	*Name.*—Mrs. R. G—.	*Age.*—24.

Description.—A Russian Pole. Dark hair and eyes; thin; nervous.

Anæsthetic used.—Nitrous oxide with 22 per cent. of air.

Operation performed.—Two teeth extracted.

Operator.—Mr. Cooper.

Notes by Mr. Bellamy Gardner.

Patient's sensations and opinions.—Thinks she remembers last tooth being removed, but is wrong as to its situation when questioned. Felt no pain. Dreamt she was talking to some one.

Remarks.—Metronome bell rings every three seconds. Some lassitude after administration. In this case the operation was purposely continued, and one tooth removed *after* patient had regained sense of hearing and power of responding to questions, but there was no pain and no distinct recollection of the removal of the tooth.

NOTE.—Where no entries made, patient quite quiet, with good breathing and good colour.

Gasometer readings.—
Before ... 84·5
After ... 32·0
―――
52·5 divisions
·18
―――
4200
525
―――
9·450 gallons used.

Duration of inhalation.	*Duration of available anæsthesia.*	*Gallons inhaled.*
150 secs.	54 secs.	9·45 gallons.

	INHALATION.	

Metronome bells.		Metronome bells.	
1		46	
2		47	
3		48	
4		49	
5		50	Administration discontinued.
6		51	
7	Responds when spoken to (" Can you hear me ? ").	52	
		53	
8		54	
9		55	
10	Breathing rather " held."	56	
11			
12			
13			
14	Responds when spoken to.		AVAILABLE ANÆSTHESIA
15			
16	Do.		
17		Metronome bells.	
18			
19	Do.		
20			
21	Do.	1	Operation begun. Patient pe fectly tranquil.
22			
23	Do.	2	
24		3	No reflex movement or noise.
25		4	
26		5	No conjunctival reflex. O tooth out.
27			
28		6	
29	Goes on moving head although not asked whether she can hear.	7	
		8	
		9	No response when spoken to.
30		10	
31		11	Perfectly tranquil. Second too grasped.
32			
33		12	
34	Breathing regular; not very deep.	13	
35		14	
36		15	Responds when spoken to. Op ration proceeding.
37			
38	Colour unaltered.	16	
39		17	Slight phonation during extra tion of tooth.
40	Pulse good and regular.		
41		18	Anæsthesia at an end.
42		19	
43		20	
44		21	
45		22	

I have chosen Case 169, as it is an interesting one. It shows that deep and tranquil anæsthesia may be obtained by nitrous oxide even when as much as 22 per cent. of air is mixed with the anæsthetic gas. And it also shows that during recovery from anæsthesia a tooth may be painlessly removed *after* the sense of hearing and the power of making responses to questions have been re-gained. The sense of hearing and the faculty of under-standing questions are late in disappearing during the inhalation of nitrous oxide gas, more especially when this gas is mixed with air, an analgesic state becoming established a considerable time before failure to hear and to respond is observed. During recovery from anæs-thesia the exact reverse obtains, the power of hearing and of understanding questions returning before the percep-tion of painful impressions is regained (see p. 172).

It would be impracticable to reproduce all the 231 cases *in extenso* as the foregoing, although they are of consider-able interest in their entirety. I have been obliged, there-fore, to condense them into the tables given on pp. 180—191.

V. The Plan by which it was found Possible to numeri-cally express the Unsatisfactory or Objectionable Phenomena of the Various Cases.

It was not until I had spent a considerable time in analysing the various cases that I devised a plan by which I could compare them one with another irrespective of the gases inhaled ; and the plan may thus be described. In certain of the cases an absolutely ideal state of anæsthesia was met with,—that is to say, the patients inhaled the gases administered to them without any discomfort, movement, or sound ; they passed into deep sleep without any alteration in colour, and without any noisy or difficult breathing ; an average dental operation was painlessly performed upon them without eliciting any phonated sounds or inconvenient reflex movements during the first thirty seconds after the

inhalation; and consciousness was regained without discomfort or disagreeable after effects. Now such cases I have regarded as having no objectionable symptoms either from the point of view of the patient, the operator, or the administrator; and I have assigned to them the value "0." In a large proportion of the cases, however, this ideal was not reached. In some it was nearly reached, and but for slight alteration in colour, or some other symptoms of minor importance, the cases were of the ideal type. I therefore assigned numbers, more or less arbitrarily fixed, to the various objectionable phenomena, and I found that I could by such a plan give a numerical expression to each and all of the objectionable phenomena met with in the various cases. The phenomena least objectionable had the fewest "bad marks;" whilst those which greatly interfered with the comfort of the patient or the convenience of the operator, or which involved any risk to the patient which was absent in other cases, had more "bad marks" assigned to them. After a while I was able to draw up the following table, which will, I hope, explain itself after what I have just said.

Table showing the arbitrary numbers chosen for expressing the objectionable phenomena met with in each administration.

		Slight.	Moderate	Extreme.
Anoxæmic convulsion	—	20	35	50
Duskiness, or change of colour	—	10	20	30
Stertor	—	5	15	40
Retching movements at end of administration or early in anæsthesia period	—	20	35	45
Phonation	—	5	15	35
Excitement and reflex movements	—	0	20	40
Forcible restraint needed	65	—	—	—
After effects	—	5	15	35
Laboured or "catchy" respiration	5	—	—	—
Unpleasant dreams	5	—	—	—
Anæsthesia after administration	Adults.	30 secs. or over. 0	25 to 29 secs. 10	Under 25 secs. 20
	Patients under 16 yrs.	25 secs. or over.	20 to 24 secs.	Under 20 secs.

By this system it will be seen that such comparisons as the following may easily be made and numerically expressed.

(1) One group of cases conducted with a certain percentage of air or oxygen may be compared with another group conducted with a different percentage, the general result of the one group being expressed by one number, and that of the other group by another number (see p. 205).

(2) The patient remaining the same, the general result of administering on one occasion a particular mixture of gases may be compared with the general result on another occasion when a different mixture is employed.

(3) A certain symptom or objectionable phenomenon may be taken, and the extent to which it is met with in certain groups of cases may be readily expressed.

For example :

(1) Taking " 0 " as the value of the ideal cases at one end of the scale, and "200" as the value ("bad marks") of the unsatisfactory cases at the other and—the latter being cases in which failure to anæsthetise took place—and working with the various empirical values given in the above table, I find that whilst the average " bad marks " of an ordinary nitrous oxide administration may be expressed by the number 93·7, those of a mixture of 5 per cent. of oxygen with nitrous oxide amount only to 22·6 ; in other words, one system of anæsthetising is four times better than the other.

(2) I found that in the case of a patient anæsthetised on one occasion with a 5 per cent. of air mixture, and on another occasion with a 5 per cent. of oxygen mixture, the average objectionable phenomena in the air administration worked out at 115, whilst those in the oxygen administration only came to 15.   Or, lastly—

(3) If we take the numbers 20, 35, and 50 to represent respectively slight, moderate, and extreme anoxæmic convulsion, we find that whilst the average degree of this convulsion in the pure nitrous oxide cases may be

expressed by the number 28·5, that in the 18 per cent. of air cases is but 19·1.

VI. The Cases of Series I.

(Comparable cases.)

For reasons already mentioned, the cases of Series I cannot be given *in extenso*. I have therefore analysed them in accordance with the plan just described, and have arranged them, under the various mixtures used, in the accompanying table.

Pure Nitrous Oxide.

No. of case.	Sex.	Age	Inhalation (secs.).	Available anaesthesia (secs.).	Gallons used.	Teeth or roots removed.	Arbitrary numerical expressions for objectionable phenomena.										Negative value of case as a whole.
							Anoxaemic convulsion.	Change of colour.	Stertor.	Retching movements.	Phonation.	Excitement or reflex movements.	After-effects.	Laboured or "catchy" respiration.	Unpleasant dreams.	Short anaesthesia.	
1	F.	19	69·	30·	5·01	3·	20·	20·	40·	...	15·	...	5·	...	5·	...	105·
2	F.	22	69·	30·	6·17	Nil	·	...	40·	...	85·	65·	...	...	...	...	140·
3	F.	24	60·	18·	3·60	1·	...	...	15·	...	85·	40·	...	...	...	20·	110·
4	F.	15	48	27·	5·86	1·	35·	20·	40·	...	...	40·	...	...	...	10·	145·
5	F.	39	60·	25·	5·63	8·	20·	20·	...	...	15·	...	...	...	...	...	65·
6	F.	37	48·	33·	5·09	2·	35·	20·	15·	...	15·	20·	...	...	...	10·	105·
7	F.	29	60·	31·	8·78	2·	...	...	40·	...	85·	40·	...	...	...	...	115·
8	F.	17	60·	26·	3·96	8·	50·	...	15·	...	...	...	...	...	...	10·	75·
9	F.	33	60·	27·	6·17	2·	85·	20·	15·	...	15·	40·	...	...	...	...	65·
18	?.	26	51·	39·	3·38	?	35·	10·	15·	...	...	20·	...	...	...	20·	105·
19	F.	18	42	15·	4·19	1·	35·	20·	15·	...	5·	...	...	...	...	...	80·
71	M.	17	57·	38·	4·44	2·	...	20·	40·	...	85·	...	...	...	...	...	70·
73	F.	25	72·	30·	5·14	2·	50·	10·	5·	...	10·	40·	...	...	...	...	65·
74	F.	39	66·	36·	3·87	1·	35·	20·	...	85·	...	20·	...	...	...	...	100·
122	F.	21	60·	45·	4·24	Nil	50·	30·	...	...	...	...	...	...	...	...	70·
123	F.	13	86·	27·	3·94	3·	50·	10·	15·	...	35·	20·	...	...	...	10·	105·
124	M.	17	60·	28·	5·23	1·	35·	30·	...	...	...	...	...	...	...	10·	125·
149	F.	19	38·	26·	3·90	2·	50·	...	15·	...	...	20·	...	...	...	10·	75·
150	F.	27	42·	42·	5·18	2·	35·	...	...	...	...	...	...	...	...	...	85·
151	M.	20	66·	45·	5·63	1·	85·	...	15·	...	...	20·	...	...	...	...	70·
Average	...	...	55·9	30·3	4·5	1·6	28·5	12·5	1·7	1·7	12·7	15·2	0·25	...	0·25	5·5	93·7

No.	Sex																
3 per cent. Air.																	
20	F.	28	60·	39·	4·23	1·	30·	10·	15·	…	…	…	…	…	5·	…	80·
21	F.	17	79·	27·	4·21	1·	35·	20·	15·	…	15·	40·	…	…	…	10·	135·
22	F.	34	57·	16·	3·43	1·	35·	10·	…	…	35·	40·	…	…	…	20·	140·
23	F.	43	76·	33·	4·98	7·	35·	20·	15·	…	…	…	…	…	…	…	70·
155	F.	35	84·	39·	8·03	1·	50·	10·	…	…	…	20·	…	…	…	…	80·
Average	…	…	60·6	30·8	4·90	2·2	35·	14·	9·	…	10·	20·	…	…	1·0	6·0	95·
5 per cent. Air.																	
15	F.	13	60·	36·	3·42	2·	50·	20·	15·	…	15·	…	…	…	…	…	70·
16	F.	17	66·	48·	4·01	2·	50·	20·	16·	…	35·	20·	…	…	…	…	100·
17	F.	24	63·	42·	5·36	1·	35·	20·	5·	…	15·	…	…	…	…	…	115·
46	M.	28	99·	42·	6·26	4·	35·	…	15·	…	…	…	…	…	…	…	65·
47	?	36	57·	33·	4·91	3·	50·	10·	…	…	…	…	…	…	…	…	60·
48	F.	32	57·	60·	4·17	6·	35·	20·	15·	…	15·	…	…	…	…	…	70·
97	F.	33	69·	24·	4·93	2·	20·	…	16·	…	16·	20·	15	…	…	…	70·
104	M.	21	87·	42·	5·49	Nil	20·	20·	15·	…	15·	…	…	…	…	20·	90·
105	M.	20	75·	38·	6·31	1·	35·	10·	5·	…	5·	…	…	…	…	…	50·
106	F.	23	80·	33·	4·86	5·	50·	20·	15·	…	15·	…	…	…	…	…	100·
Average	…	…	69·3	39·9	5·05	2·6	38·	11·	10·	…	0·6	4·	1·5	…	…	2·0	79·
6 per cent. Air.																	
24	F.	20	69·	36·	3·76	4·	35·	10·	5·	…	…	20·	…	…	…	…	70·
25	F.	14	45·	30·	4·41	1·	…	30·	15·	…	15·	20·	…	…	5·	…	70·
26	F.	10	51·	30·	2·73	2·	35·	20·	15·	…	15·	40·	…	…	…	…	125·
27	F.	21	63·	51·	4·91	4·	20·	20·	15·	…	15·	…	…	…	…	…	70·
159	F.	36	60·	33·	5·97	1·	20·	20·	15·	…	15·	20·	…	…	…	…	90·
160	M.	15	69·	34·	6·64	3·	20·	20·	15·	…	…	…	…	…	…	…	55·
Average	…	…	59·5	35·6	4·73	9·5	21·6	20·	12·3	…	7·5	10·6	…	…	5	…	80·

No. of case.	Sex	Age	Inhalation (secs).	Available anæsthesia (secs.)	Gallons used	Teeth or roots removed.	Anæsthetic convulsion	Change of colour.	Stertor.	Retching movements.	Phonation.	Excitement or reflex movements.	After-effects.	Laboured or "catchy" respiration.	Unpleasant dreams.	Short anæsthesia.	Negative value of case as a whole.
								Arbitrary numerical expressions for objectionable phenomena.									
7 per cent. Air.																	
28	F.	12	45·	30·	3·04	2·	35·	10·	40·	...	...	20·	...	...	...	...	105·
29	F.	22	60·	30·	4·10	1·	35·	20·	15·	...	...	40·	...	...	...	...	110·
30	M.	17	60·	36·	5·84	Nil	35·	10·	15·	...	...	20·	...	...	...	...	80·
31	F.	30	72·	33·	5·32	1·	35·	10·	...	...	15·	...	...	...	...	...	45·
161	F.	21	60·	30·	5·07	2·	20·	40·	15·	...	...	...	...	...	...	...	75·
162	M.	14	60·	46·	5·56	2·	20·	10·	15·	...	...	...	...	...	...	...	45·
Average	...	...	59·5	34·1	4·73	1·2	30	16·6	14·1	...	2·5	13·3	...	...	...	...	76·6
10 per cent. Air.																	
34	F.	38	75·	33·	6·24	6·	35·	20·	15·	...	...	...	...	...	...	...	70·
35	F.	20	63·	21·	5·84	1·	35·	...	15·	...	15·	40·	...	...	5·	20·	130·
36	F.	28	87·	36·	4·93	1·	20·	10·	15·	...	...	...	...	...	...	...	45·
37	F.	20	63·	54·	5·22	3·	20·	...	15·	...	35·	...	...	...	...	...	35·
55	M.	29	81·	30·	5·88	5·	35·	10·	15·	...	...	...	5·	...	...	...	100·
56	F.	28	66·	30·	5·05	2·	35·	...	...	...	...	...	...	...	...	...	35·
100	F.	31	123·	48·	11·01	1·	35·	10·	...	...	...	...	...	...	...	...	45·
101	F.	21	89·	18·	9·98	1·	85·	...	15·	...	15·	20·	...	...	...	20·	90·
102	F.	48	98·	39·	9·41	2·	...	10·	15·	...	15·	20·	...	...	...	...	60·
103	F.	33	81·	46·	6·62	Nil	85·	20·	...	...	15·	20·	...	...	...	...	90·
Average	...	...	80·4	35·5	6·71	2·9	29·5	8·	9·	...	9·6	10·	·5	...	·5	4·	70·

Arbitrary numerical expressions for objectionable phenomena.

No. of Case	Sex	Age	Inhalation (secs.)	Available anaesthesia (secs)	Gallons used	Teeth or roots removed	Anoxaemic convulsion	Change of colour	Stertor	Retching movements	Phonation	Excitement or reflex movements	After-effects	Laboured or "catchy" respiration	Unpleasant dreams	Short anaesthesia	Negative value of case as a whole
18 per cent. Air.																	
43	—	23	69·	45·	5·63	1·	85·	10·	15·	…	15·	…	5·	…	…	…	80·
44	F.	20	84·	48·	4·03	3·	20·	…	15·	…	…	…	5·	…	…	…	40·
45	F.	32	87·	45·	6·12	7·	20·	20·	15·	…	15·	20·	…	…	…	…	70·
49	F.	30	129·	42·	8·53	4·	2?·	20·	5·	…	…	…	…	…	5·	…	70·
50	F.	25	114·	30·	9·10	2·	20·	20·	15·	…	15·	…	…	…	…	…	70·
Average …		…	96·6	42·	6·6	3·4	23·	14·	13·	…	9·	4·	2·	…	1·	…	·66
18 per cent. Air.																	
83	F.	24	147·	30·	6·87	1·	20·	10·	…	…	…	…	…	…	…	20·	30·
84	F.	22	00·	21·	4·59	2·	35·	10·	15·	…	35·	…	…	…	…	10·	115·
85	F.	20	84·	26·	?	3·	…	20·	…	…	35·	40·	…	…	…	…	65·
87	F.	23	90·	30·	5·71	2·	20·	10·	5·	…	15·	…	…	…	5·	…	95·
88	M.	14	102·	42·	6·62	2·	20·	10·	15·	…	…	…	…	…	…	…	45·
89	M.	31	141·	54·	10·31	3·	20·	10·	…	…	…	…	…	…	…	…	30·
90	F.	13	81·	39·	5·16	2·	20·	10·	…	…	…	20·	…	…	…	…	30·
91	F.	29	69·	32·	6·42	3·	20·	10·	…	…	…	40·	…	…	…	10·	60·
92	F.	49	87·	30·	8·82	4·	20·	10·	15·	…	35·	…	…	…	…	…	80·
93	F.	28	99·	27·	8·82	8·	…	…	…	…	…	…	…	…	…	…	100·
95	F.	28	111·	46·	7·77	5·	35·	10·	15·	…	35·	…	…	…	…	10·	45·
96	F.	35	144·	25·	11·98	3·	20·	20·	5·	…	5·	20·	…	…	…	…	80·
Average …		…	108·7	33·5	7·3	2·7	19·1	10·8	4·5	…	10·4	10·	…	…	·4	4·1	69·6

20 per cent. Air.

No.	Sex & age															
51	F. 22	120·	42·	7·32	2·	..	10·	5·	..	..	..	5·	..	..	..	20·
52	F. 46	126·	42·	9·77	4·	..	20·	..	..	5·	..	..	..	..	..	25·
53	F. 22	96·	86·	5·83	2·	35·	..	15·	..	35·	40·	..	..	5·	..	130·
54	F. 23	87·	33·	7·07	3·	..	20·	..	..	35·	20·	..	..	..	..	75·
66	M. 19	99·	36·	5·83	2·	..	20·	15·	..	35·	20·	..	..	..	..	90·
67	M. 22	78·	33·	8·08	2·	35·	20·	..	..	..	..	..	..	..	..	55·
82	F. 22	111·	30·	13·03	2·	20·	10·	5·	..	15·	20·	..	..	..	..	70·
Average	...	102·4	36·	8·1	2·4	12·8	14·2	5·7	..	17·8	14·2	0·7	·3	7·	..	66·4

22 per cent. Air.

No.	Sex & age															
109	F. 23	162·	36·	9·63	10·	..	..	..	..	5·	..	5·	..	..	..	10·
110	F. 13	123·	30·	8·01	2·	..	10·	..	..	..	..	..	5·	..	..	10·
168	F. 21	135·	42·	11·41	?	..	..	..	..	..	..	5·	..	..	..	5·
169	F. 24	150·	54·	9·45	2·	..	10·	..	..	5·	..	..	..	..	..	5·
170	F. 18	147·	63·	11·17	3·	..	10·	..	..	35·	20·	..	..	..	10·	30·
195	F. 19	117·	27·	8·96	2·	20·	..	..	..	..	..	..	..	..	10·	75·
196	F. 12	99·	27·	6·51	3·	20·	20·	..	..	15·	..	..	..	..	..	30·
208	M. 30	219·	57·	13·37	3·	20·	10·	..	..	..	20·	..	..	..	..	75·
209	M. 14	105·	54·	5·20	2·	20·	20·	..	..	..	..	..	..	..	..	30·
210	F. 20	144·	45·	9·30	2·	..	..	..	..	..	20·	..	..	..	..	25·
211	F. 22	93·	42·	7·00	5·	35·	20·	5·	..	..	40·	..	..	5·	10·	55·
212	M. 15	117·	27·	5·92	1·	20·	10·	..	..	5·	..	..	..	5·	..	90·
213	F. 17	129·	45·	4·80	3·	20·	10·	5·	..	..	..	..	..	5·	..	35·
214	F. 29	150·	48·	12·67	4·	20·	10·	5·	..	..	..	..	..	..	..	40·
Average	...	135·	42·6	8·81	3·2	12·5	8·5	1·	..	4·2	7·1	0·7	·3	..	2·1	36·7

¹ Bag occasionally distended during inhalation.

Arbitrary numerical expressions for objectionable phenomena.

No. of case.	Sex.	Age.	Inhalation (secs.).	Available anaesthesia (secs.).	Gallons used.	Teeth or roots removed	Anoxæmic convulsion.	Change of colour.	Stertor.	Retching movements.	Phonation.	Excitement or reflex movements.	After effects.	Laboured or "catchy" respiration.	Unpleasant dreams.	Short anæsthesia.	Negative value of case as a whole.
25 per cent. Air.																	
65	F.	13	120·	45·	5·18	2·	...	10·	15·	...	...	...	...	...	...	...	25·
68	F.	26	102·	39·	9·99	3·	35·	10·	15·	...	5·	...	...	...	...	...	65·
69	F.	18	99·	33·	7·65	2·	20·	...	...	...	5·	...	5·	...	...	10·	10·
72	F.	8	93·	27·	4·24	2·	...	10·	5·	...	65·	...	...	...	5·	20·	60·
75	M.	26	103	38·	17·64¹	3·	20·	...	...	...	35·	...	...	...	...	...	60·
79	F.	22	138·	36·	17·64¹	4·	35·	10·	...	...	35·	...	...	...	...	...	65·
80	F.	31	111·	32·	15·57¹	Nil		20·	...	...	35·	65·	...	...	...	...	155·
111	F.	26	126·	about 51·	10·63	2·	20	...	...	...	...	...	5·	...	...	...	25·
Average	...	...	111·3	37·	7·53	2·2	16·2	7·5	4·3	...	16·2	8·1	1·2	...	·6	3·7	58·1
30 per cent. Air.																	
76	F.	24	129·	32·	12·0	5·	...	10·	...	...	15·	...	15·	...	...	...	26·
77	F.	35	127·	34·	11·34¹	4·	...	...	...	...	...	...	...	...	...	...	15·
78	F.	28	141·	14·	17·64¹	2·	...	10·	...	...	35·	40·	...	...	...	20·	105·
171	F.	24	195·	38·	15·26	2·	...	...	...	35·	15·	40·	...	...	...	...	90·
Average	...	...	148·	29·7	...	3·2	0·	5·	...	8·7	16·2	20·	3·7	...	...	5	53·7
33⅓ per cent. Air.																	
127	F.	35	279·	Nil	17·64	?						20·					200·
												Failure to produce anaesthesia.					
3 per cent. Oxygen																	
61	M.	29	72·	?	9·10	Nil	35·	10·	15·	...	...	...	...	...	...	...	60·
62	M.	13	81·	46·	5·27	2·	50·	10·	15·	...	...	...	...	...	...	...	75·
145	F.	32	120·	45·	8·76	?	20·	20·	...	...	15·	...	...	...	...	...	55·
146	F.	39	102·	45·	8·87	Nil	30·	20·	...	...	15·	20·	...	...	...	...	75·
158	F.	33	108·	51·	8·64	6·	...	10·	...	...	...	...	...	...	...	...	10·
Average	...	...	96·6	46·5	8·12	3·	25·	14·	6·	...	6·	4·	...	...	...	...	55·

4 per cent. Oxygen.

No.	Sex	Age															
12	F.	9	60·	36·	2·73	2·	35·	10·	15·								60·
13	F.	24	90·	57·	7·50	4·	20·	10·	5·								35·
14	F.	9	69·	42·	5·43	2·	20·	10·			20·				5·		85·
57	F.	31	138·	54·	6·15	6·		20·	5·								40·
58	F.	19	129·	63·	6·60	6·		20·	5·			5·					30·
132	M.	28	129·	45·	11·21	6·	20·	20·				15·					60·
143	F.	23	105·	42·	9·41	7·	20·	10·				15·					45·
144	F.	42	81·	27 or more	8·22	3·			15·						5·	10·	10·
156	M.	30	111·	41·	9·72	1·	35·		15·			5·					55·
157	F.	22	90·	57·	6·87	6·	20·	10·					5·				35·
Average		...	101·1	46·4	7·38	4·3	17·	11·	4·5		4·	2·	1·4		·5	1·	40·5

5 per cent. Oxygen.

No.	Sex	Age															
32	F.	25	105·	39·	8·46	1·		10·									15·
33	F.	21	105·	40·	6·69	2·	20·	10·	15·					5·			45·
112	F.	15	132·	27·	5·56	1·		10·					·5				15·
113	M.	18	96·	42·	10·27	2·		10·				5·					10·
125	M.	21	138·	42·	10·27	4·		10·				5·					10·
126	F.	46	93·	36·	7·36	2·						5·			5·		15·
128	F.	24	135·	81·	14·40	4·		10·									5·
130	M.	22	129·	42·	9·00	4·											25·
131	M.	32	144·	45·	9·16	3·		10·									15·
206	F.	21	141·	37·	6·26	3·		10·	15·		20·					10·	0·
207	F.	38	159·	45·	6·03	4·	20·	10·						5·	20·		45·
217	M.	22	126·	54·	7·86	1·		10·	15·			5·					40·
218	F.	21	120·	30·	5·43	1·		10·						5·			25·
219	F.	33	138·	54·	8·04	1·	20·	20·	15·		20·				20·	10·	5·
220	F.	27	138·	27·	6·33	2·		10·									90·
224	M.	16	138·	54·	11·84	1·		10·									10·
225	M.	12	87·	48·	5·11	1·		10·	5·								15·
Average		...	124·9	43·7	8·12	2·1	3·5	8·8	2·05		2·6	2·3	1·4		11	·5	22·6

No. of case	Sex	Age	Inhalation (secs.)	Available anæsthesia (secs.)	Gallons used	Teeth or roots removed	Anoxæmic convulsions	Change of colour	Snores	Retching movements	Phonation	Excitement or reflex movements	After effects	Laboured or choky respiration	Unpleasant dreams	Short anæsthesia	Negative value of this as a whole
6 per cent. Oxygen																	
189	F.	23	144·	60·	?	4·	…	10·	…	…	…	85·	…	…	…	…	85·
135	M.	25	162·	45·	8·10	2·	…	10·	…	…	…	…	…	…	…	…	10·
186	F.	18	120·	86·	7·72	2·	…	…	…	90·	…	…	…	…	…	…	90·
137	M.	17	128·	51·	9·75	1·	…	10·	…	…	…	…	…	…	…	…	10·
138	F.	31	180·	51·	7·88	?	…	…	…	…	…	…	…	…	…	…	0·
139	F.	26	99·	38·	7·83	5·	…	10·	…	…	15·	…	…	…	…	…	15·
140	M.	29	99·	54·	9·81	2·	…	20·	15·	…	15·	…	…	…	…	…	10·
141	F.	23	108·	86·	7·88	1·	…	10·	…	40·	15·	…	…	…	…	…	100·
148	F.	35	123·	83·	0·75	2·	…	90·	…	…	85·	…	…	5·	…	…	90·
147	M.	28	185·	86·	8·28	1·	…	10·	…	…	…	…	…	5·	…	…	70·
148	F.	30	129·	28·	9·86	1·	…	10·	…	…	…	…	…	…	…	10·	55·
Average	…	…	139·5	42·	8·67	2·1	…	8·1	1·8	8·1	7·8	5·4	·9	…	…	·9	577·
7 per cent. Oxygen																	
10	F.	14	135·	42·	7·25	4·	…	…	15·	90·	15·	90·	…	…	…	…	50·
11	F.	13	99·	42·	?	2·	…	10·	…	…	…	…	…	…	…	…	0·
63	F.	26	144·	57·	7·07	3·	…	10·	5·	…	…	…	…	…	…	…	90·
64	F.	16	117·	60·	6·31	4·	…	10·	5·	…	…	…	…	…	…	…	15·
152	F.	25	147·	63·	10·78	4·	…	10·	5·	…	…	90·	15·	…	…	…	15·
158	M.	16	141·	46·	7·29	3·	…	10·	…	…	…	90·	…	…	…	…	90·
154	F.	86	87·	45·	8·10	5·	…	…	5·	…	…	…	…	…	…	…	0·
172	F.	14	128·	48·	7·81	8·	…	10·	…	…	…	…	…	…	…	…	90·
178	F.	15	141·	51·	8·28	2·	…	…	…	…	…	…	…	…	…	…	0·
174	F.	28	159·	51·	10·02	?	…	10·	5·	…	…	…	5·	…	5·	…	85·
175	F.	28	162·	48·	10·8	?	…	…	…	…	…	…	…	…	…	…	0·
Average	…	…	132·6	50·1	8·54	3·3	…	5·4	1·8	…	5·7	8·4	·5	…	·5	…	177·

8 per cent. Oxygen																	
114	F.	39	135·	48·	9·46	8·											0·
115	F.	33	117·	42·	8·17	6·						·5				5·	5·
176	F.	19	162·	42·	10·32	3·				20·							20·
177	F.	23	150·	48·	10·47	1·											30·
178	M.	32	177·	30·	13·14	1·	10·	10·			15·			5·			65·
179	F.	30	162·	39·	11·66	1·					15·		40·				40·
180	F.	12	129·	33·	6·22	2·				20·			20·				0·
181	M.	24	129·	33·	14·05	3·					15·		65·				100·
182	F.	23	144·	45·	10·99	4·					35·	5·					40·
183	F.	25	174·	50·	10·81	7·						5·	20·				25·
184	F.	19	207·	60·	15·66	4·											0·
187	F.	21	159·	39·	8·76	8·											0·
188	F.	35	129·	42·	8·87	2·											0·
215	F.	36	139·	38·	6·84	2·				20·	5·		20·				0·
216	F.	24	132·	57·	6·26	4·					5·						25·
221	F.	26	159·	86·	9·75	4·					5·		20·			5·	5·
222	F.	21	120·	33·	8·40	2·											35·
223	F.	24	123·	45·	9·23	10·								10·			10·
Average			147·	41·0	9·94	3·7	···	1·1	·5	2·2	5·2	11·3	·2	···	·2	·5	21·6

9 per cent. Oxygen																	
185	M.	15	141·	57·	8·06	2·			5·								10·
186	F.	23	182·	38·	9·54	7·							5·		5·		5·
190	M.	21	171·	42·	11·01	2·			5·								0·
191	F.	21	117·	54·	6·6)	1·					15·	40·					5·
192	M.	23	177·	39·	12·83	3·											55·
Average			147·6	45·	9·60	2·	···	···	2·	···	3·	8·	1·	···	1·	···	15·

1 Bag occasionally distended during inhalation.

No. of case.	Sex.	Age.	Inhalation (secs.).	Available anaesthesia (secs.).	Gallons used	Teeth or roots removed.	Anoxæmic convulsion.	Change of colour.	Stertor.	Retching movements.	Phonation.	Excitement or reflex movements.	After effects.	Laboured or "catchy" respiration.	Unpleasant dreams.	Short anaesthesia.	Negative value of case as a whole.
							Arbitrary numerical expressions for objectionable phenomena.										
10 per cent. Oxygen.																	
30	F.	33	138·	45·	10·27	7·	...	10·	...	...	...	...	...	...	...	...	0·
116	F.	29	132·	63·	9·81	5·	...	...	...	...	...	...	...	...	5·	...	15·
117	F.	85	123·	42·	7·88	5·	...	...	...	...	15	...	5·	...	...	...	6·
118	F.	?	108·	57·	7·70	4·	...	...	...	...	...	...	...	...	...	...	15·
121	F.	31	270·	54·	?	3·	...	...	...	...	...	...	...	...	...	...	0·
180	F.	19	174·	51·	?	0·	...	...	...	...	...	40·	...	...	...	...	40·
108	F.	25	138·	37·	6·51	8·	...	...	...	...	15·	40·	...	...	...	...	40·
194	M.	12	114·	30·	4·87	3·	...	...	15·	...	...	40·	...	...	...	...	70·
197	F.	81	168·	42·	7·48	1·	...	...	...	...	·5	...	...	...	...	...	0·
198	F.	18	177·	33·	8·64¹	1·	...	...	...	...	...	40·	...	...	...	...	45·
Average ...			155·1	45·4	7·78	3 3	...	1·	1·6	...	8·5	16·	·5	...	·5	...	28·
11 per cent. Oxygen																	
199	F.	43	188·	54·	10·65	5·	...	...	...	...	5·	...	5·	...	...	...	15·
200	F.	21	147·	33·	5·92	4·	...	...	...	...	...	20·	5·	...	5·	...	25·
201	F.	18	192·	39·	8·13	1·	...	...	...	...	15·	20·	15·	...	...	...	20·
202	F.	37	255·	70·	9·03	4·	...	...	15·	...	5·	40·	15·	...	...	...	45·
203	F.	11	171·	24·	4·51	?·	...	...	...	...	...	...	...	5·	...	10·	105·
204	F.	21	126·	45·	13·30¹	3·	...	...	...	35·	...	...	15·	...	...	...	5·
205	F.	13	153·	36·	11·70¹	2·	...	...	...	...	...	...	...	...	...	...	15·
Average ...			175·9	43·	7·64	3·3	...		2·1	5·	3·5	11·4	7·8	·7	·7	1·4	33·3

13 per cent. Oxygen.															
59	F.	29	174·	36·	9·32	7·	...	...	...	...	...	20·	...	...	...	20·
60	?	54	150·	42·	5·90	2·	...	...	...	...	..5·	·	...	...	...	5·
20 per cent. Oxygen.															
226	F.	11	228·	39·	8·87	2·	...	...	...	...	...	...	...	...	...	0·
227	F.	42	192·	27·	12·60	3·	...	...	35·	35·	35·	40·	...	...	10·	85·
228	F.	24	228·	57·	11·1	5·	...	...	...	...	15·	...	...	5·	...	40·
229	F.	32	246·	36·	10·9	3·	...	...	...	...	...	40·	...	...	...	60·
Average ...	...	...	223·5	39·7	10·86	3·2	...	...	...	8·7	12·5	20·	1·2	1·2	2·5	46·2

[1] Bag occasionally distended during inhalation.

VII. A Comparative Study of the Effects produced by the Administration of the Different Gaseous Mixtures.

1. *The duration of inhalation which was found to be necessary for the production of anæsthesia for a short dental operation.*—In the accompanying chart the average duration in each group of cases is given. In every administration the inhalation was continued till, from the character of the breathing, the insensibility of the conjunctiva, or other conditions, it was clear that anæsthesia had been obtained.

Chart I.—*Showing average duration of inhalation necessary for the production of anæsthesia for a dental operation.*

It will be seen that the shortest average inhalation was recorded with pure nitrous oxide (55·9 secs.), which means that when this gas is administered without any oxygen asphyxial symptoms come on within a minute, and prevent the intake of more of the anæsthetic gas. In many cases the anoxæmic phenomena come about before anæsthesia is fully established. This is notably the case in persons with enlarged tonsils, post-nasal adenoid growths, or other obstructive affections of the

upper respiratory passages. It will be seen that as the proportion of air increases there is an increasing tendency, with one or two irregular fluctuations due to accidental circumstances, for the inhalation period to increase, till with 30 per cent. of air it reaches 148 secs. The same tendency is shown, and far more markedly, in the case of mixtures containing oxygen and no nitrogen. With 8 per cent. of oxygen the available inhalation period was 96·6 secs.; and with 20 per cent. of oxygen it was 223·5 secs.

2. *Average duration of anæsthesia after inhalation.*— In operations within and about the mouth, nose, or pharynx, the length of time during which a patient will

CHART II.—*Showing average duration of available anæsthesia after administration.*
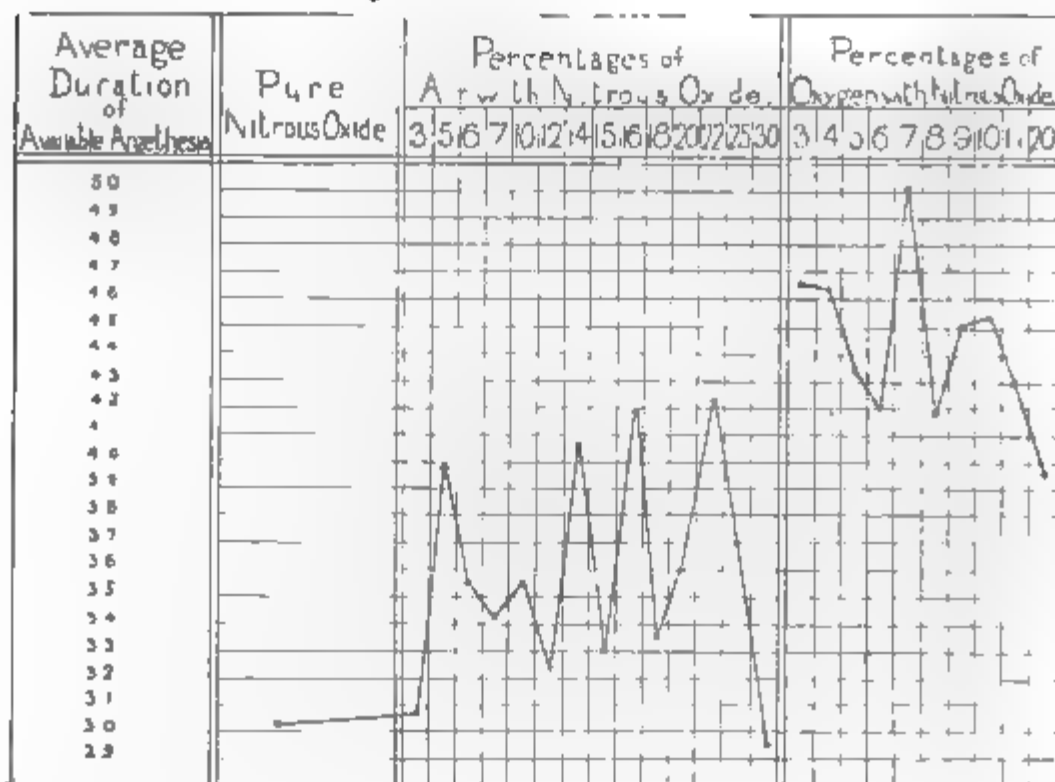
remain anæsthetised after the removal of the face-piece becomes a matter of importance. The expression "available anæsthesia" is often employed to designate this after-insensibility. The most noticeable feature in the accompanying chart is that the available anæsthesia which results from the use of nitrous oxide mixed with

oxygen is very distinctly longer than that from nitrous oxide mixed with air.

It is true that in both sets of administrations the variations are considerable, being dependent upon other factors than the actual percentages of air or oxygen used. With pure nitrous oxide the average anæsthesia was 80·3 secs. In the air cases the shortest average anæsthesia was met with when employing 3 per cent. of air (30·8 secs.) and 30 per cent. of air (29·7 secs.), i. e. at the two extremes; the longest average anæsthesia being recorded with 14 per cent., 16 per cent., and 22 per cent. of air, the respective durations with these percentages being 40·5 secs., 42 secs., and 42·6 secs. With regard to the oxygen cases it will be seen that the lowest average anæsthesia (39·7) was very little below the highest (42·6) of the air cases. The best results, so far as a lengthy available anæsthesia is concerned, were met with when using 7 per cent. of oxygen, the average duration of anæsthesia after inhaling nitrous oxide mixed with this percentage of oxygen being 50·1 secs.

CHART III.—*Showing average quantity of gas or gases inhaled for the production of anæsthesia for a dental operation.*
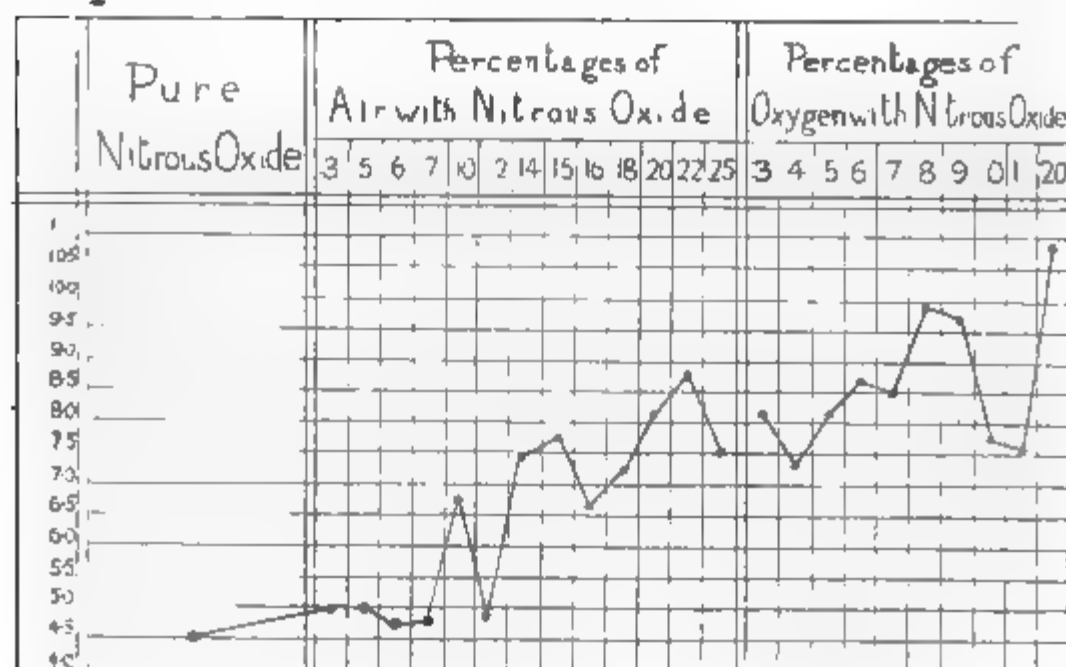
3. *Average quantity of nitrous oxide or its mixtures needed to produce anæsthesia for a short dental operation.* —There is very little to be said concerning the accompanying chart, which, as one would expect, closely resembles that giving the duration of the inhalation. The slight want of correspondence between the two charts is probably owing to the occurrence of apnœic pauses in breathing in many of the cases.

4. *Anoxæmic convulsion during the inhalation or immediately after.*—This chart is, perhaps, the most interesting of the series, at all events from a physiological point of view. It clearly shows that the convulsive

CHART IV.—*Showing average degree of anoxæmic convulsion.*

Arbitrary Numerical Scale.—None = 0; slight = 20; moderate = 35; extreme = 50.
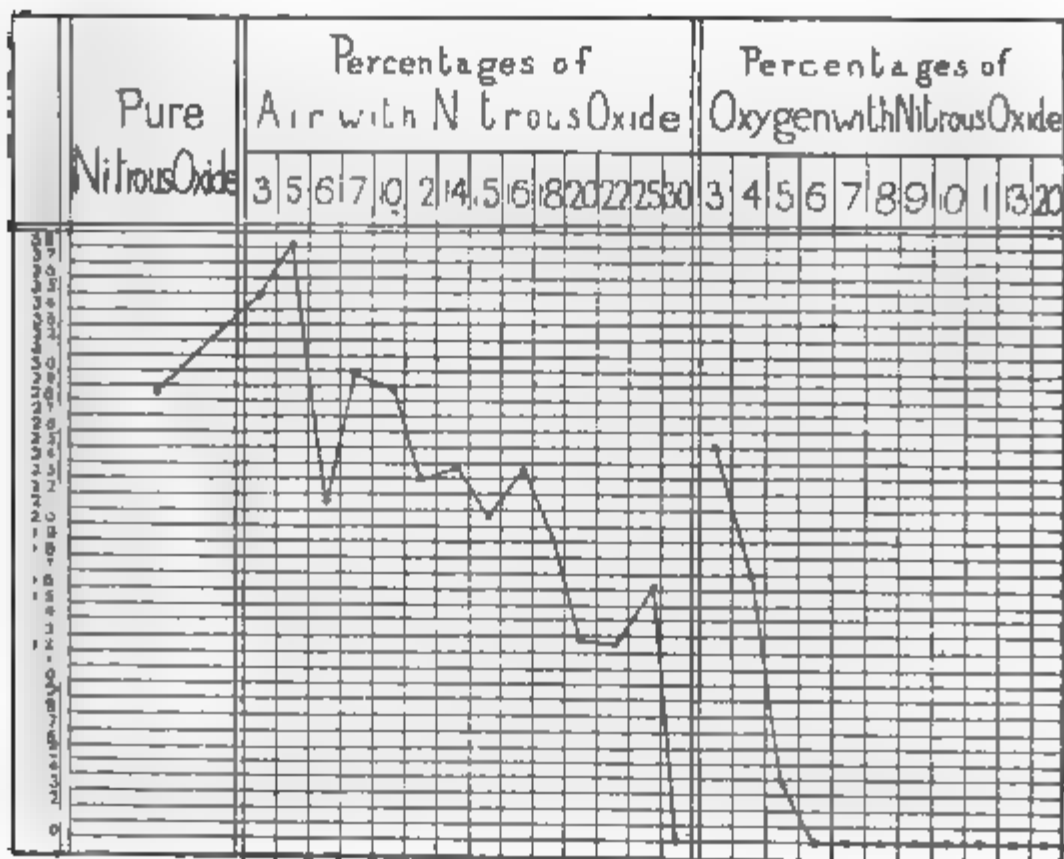
muscular phenomena with which we are so familiar when employing nitrous oxide free from oxygen are directly dependent upon the absence of this latter gas.

During the inhalation of nitrous oxide, either pure or

with percentages of air up to 25 or 30 per cent., or with oxygen up to 5 per cent., some degree of anoxæmic convulsion is very common. Roughly speaking, the more the mixture approaches to pure nitrous oxide—in other words, the greater the degree to which free oxygen is excluded—the greater will be the tendency for anoxæmic convulsion to arise ; and conversely, the greater the proportion of oxygen mixed with nitrous oxide, either as air or as the pure gas, the less will that tendency become. When free oxygen is completely or almost completely absent the patient may be thrown into the most violent epileptiform state, the whole body participating in the attack. Some patients display an unusual susceptibility to this condition, whilst others may inhale large quantities of pure nitrous oxide before any convulsive phenomena appear. Anæmic subjects, alcoholic patients, and those with any pre-existing respiratory affection are more susceptible than others. By referring to the chart it will be seen that with the larger percentages of air and of oxygen the convulsive movements are but slightly marked. In some of these slighter cases the facial muscles seem to be chiefly affected by the convulsive seizure ; in others the whole body may mildly oscillate, the convulsive spasm apparently chiefly affecting the trunk muscles ; in others the arms, legs, and hands alone may twitch ; whilst in a fourth group of cases the neck may be affected by barely perceptible clonic spasm, so that the head of the patient is felt by the administrator to move with fine rhythmic jerks in one or other direction. The anoxæmic convulsion of pure nitrous oxide becomes progressively attenuated and weakened, so to speak, as the proportion of oxygen mixed with the anæsthetic gas increases. It is curious to note in the accompanying chart an apparent contradiction to this statement. It will be observed that rather more convulsive movement was recorded with 3 and 5 per cent. of air than with nitrous oxide alone. The explanation of this is that the obstructive stertor which rapidly comes on in a pure

nitrous oxide inhalation often cuts short the administration before convulsive movements have had time, as it were, to make their appearance. The marked degree of stertor with nitrous oxide free from air or oxygen is seen in the stertor chart. The chart now under consideration shows that the greatest degree of anoxæmic convulsion was met with when nitrous oxide was given with 3 per cent. or 5 per cent. of air. As the proportion of air increases, the convulsive movements progressively lessen, till, when 30 per cent. of air is administered with nitrous oxide they cease to appear. The decline in anoxæmic convulsion is, for obvious reasons, more marked in the oxygen cases, the symptom altogether vanishing when this gas is present to the extent of 6 per cent. or more.

In preparing the above chart no attention was paid to any convulsive phenomena which were noted during the operation, the reason for this omission being that during the extraction of teeth operators frequently depress the lower jaw, so that the breathing becomes temporarily obstructed, and convulsive phenomena of a somewhat different origin from those under consideration may therefore become developed.

5. *Alteration in colour during inhalation or immediately after*.—Like other charts, the accompanying one shows very clearly the fitful and irregular results met with in the air cases, and the more reliable and regular effects obtainable when employing oxygen. It will be observed that a somewhat greater degree of lividity was recorded with small percentages of air than with pure nitrous oxide, and the explanation of this apparent anomaly is doubtless the same as that to which I referred when dealing with anoxæmic convulsion, viz. that the obstructive stertor which comes on so quickly with pure nitrous oxide necessitates the withdrawal of the anæsthetic before convulsive phenomena and cyanosis have had time, so to speak, to become pronounced. It will be observed that as much as, or more than 11 per cent. of oxygen is needed to prevent all alteration in colour, a point which

CHART V.—*Showing average degree of alteration in colour.*

Arbitrary Numerical Scale.—No alteration = 0; slight = 10; moderate = 20; extreme = 30.

	Pure Nitrous Oxide	Percentages of Air with Nitrous Oxide.																Percentages of Oxygen with Nitrous Oxide											
		3	5	6	7	10	12	14	15	16	18	20	22	25	30	3	4	5	6	7	8	9	10	11	13	20	
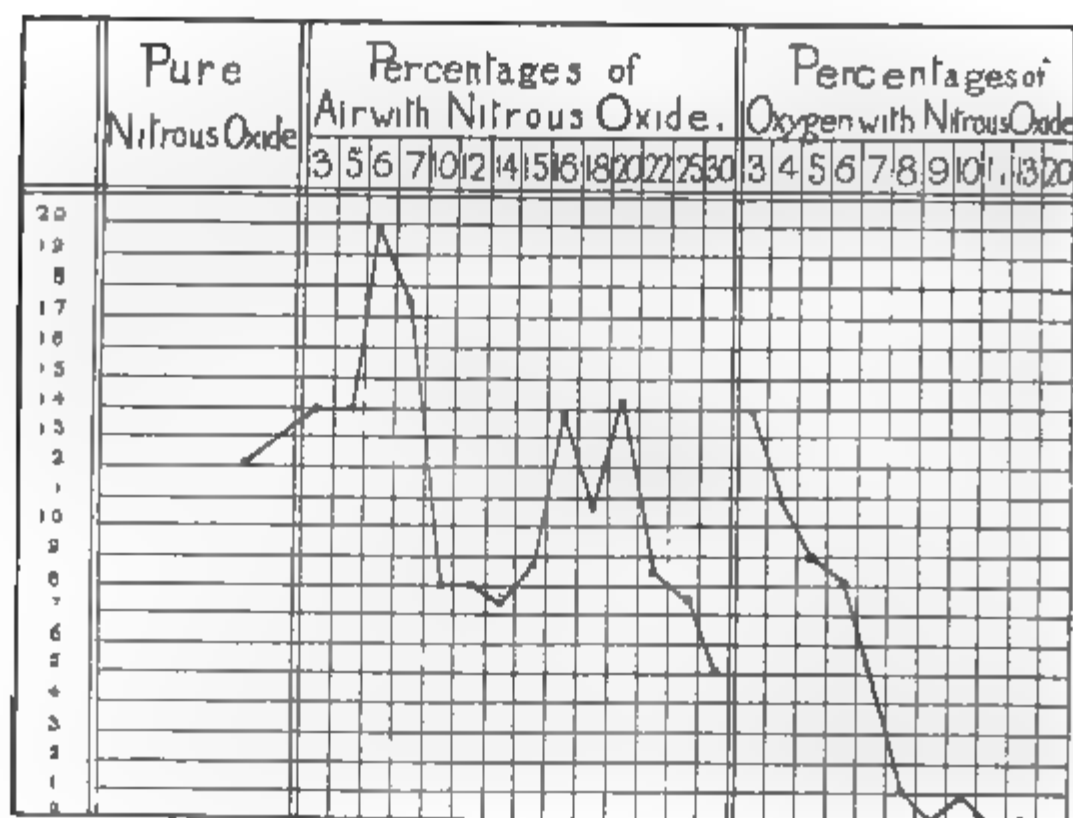
is of interest, because it proves that in administering nitrous oxide and oxygen we must not endeavour to keep the patient's colour perfectly normal in every case, otherwise we may be administering such a percentage of oxygen as may interfere with proper anæsthesia. Except in the case of children and weakly women, mixtures of nitrous oxide with 11 per cent. of oxygen are liable to lead to intoxication-effects, rather than to deep anæsthesia (see p. 203).

In drawing up the above chart no attention was paid to recorded alterations in colour during the operation period, as such alterations are often dependent upon intercurrent mechanical causes.

6. *Stertor during inhalation.*—The respiratory changes under nitrous oxide and its mixtures are of considerable interest. Under the influence of the pure gas breathing becomes increasingly deeper and quicker, till, at the end

of about 55 or 60 secs., anoxæmic convulsion and obstruc-
tive stertor supervene, and the inhalation has to be
suspended. In some cases convulsion predominates; in
others stertor is more conspicuous. Either, however, is
capable of interfering with respiratory rhythm, and of
arresting breathing if the administration be pushed suffi-
ciently far.

Three varieties of stertor may be met with under pure
nitrous oxide, viz. (1) the so-called "false" stertor—
loud, coarse, regular, and coming on before true anæs-
thesia is established; (2) a softer, snoring sound, identical
in its character, and possibly also in its immediate causa-
tion, with the usual stertor of ether or chloroform narcosis;
and (3) the sharp, loud, irregular, and deeply seated
stertor of an obstructive and choking type, so character-
istic of pure nitrous oxide when administered to its full
extent.

Little need be said as to the first variety. It is most
liable to arise in a patient with an elongated or enlarged
uvula; it is apparently due to the soft palate vibrating
against the pharynx; and it occurs independently of the
depth of anæsthesia present.

The two kinds of true stertor are, however, more im-
portant. True stertor may become audible in a mild
form about two thirds of the way through the administra-
tion, and it then has as its immediate cause vibration of
the base of the tongue against the pharyngeal wall. As
the administration proceeds the softly snoring breathing
gradually, or possibly suddenly, becomes replaced by the
coarse and obstructive stertor; that is to say, the first
variety of true stertor passes into the second.

Respiratory rhythm is not interfered with by simple
snoring, although the latter invariably causes the breath-
ing to increase in rate and depth. When, however, deep
and obstructive stertor sets in, respiratory rhythm is at
once altered, the alteration bearing a direct relation to
the degree of obstruction. The coarse and irregular
stertor so common at the acme of an administration of

pure nitrous oxide seems to be immediately dependent, in great measure at all events, upon spasmodic elevations of the whole larynx, the upper aperture of which becomes intermittently occluded by the epiglottis. Each time the larynx ascends to meet the epiglottis the air-way closes, and it is this closure which produces the stertor and the altered rhythm of respiration. It is most probable that the spasm which thus raises and lowers the larynx when pure nitrous oxide is administered to its fullest extent is, like the other muscular movements already considered, anoxæmic in its nature. In some cases of deep nitrous oxide stertor the sound is no doubt chiefly dependent upon the swollen tongue intermittently meeting the pharyngeal wall, but the elevation of the larynx here described is certainly a factor in most instances.

When air or oxygen is mixed in moderate proportions with nitrous oxide the breathing does not become as quick and deep as with the pure gas (unless emotional influences come into play); there is not that tendency for forcible expiration to become a conspicuous feature of the administration; and stertor is either present in a mild and non-obstructive form or is absent altogether.

A plentiful supply of oxygen with nitrous oxide leads either to peaceful, sleep-like, and barely perceptible breathing, or to complete arrest of respiratory movements (" physiological apnœa "). The apnœic state thus induced is distinguished from paralytic cessation of breathing by the presence of a good pulse and colour. " Physiological apnœa " never arises under pure nitrous oxide or under mixtures poor in oxygen. It is, moreover, almost invariably preceded by a phase of rapid breathing, the large proportion of oxygen favouring the development of rapid (emotional) respiration.

The most conspicuous feature of the accompanying chart is that the deepest stertor was met with when nitrous oxide was administered free from air or oxygen.

Although there are considerable fluctuations in the air cases, the gradual tendency for stertor to subside as the

CHART VI.—*Showing average degree of stertor.*

Arbitrary Numerical Scale.—None = 0; slight = 5; moderate = 15; extreme = 40.

|	Pure Nitrous Oxide	Percentages of Air with Nitrous Oxide													Percentages of Oxygen with Nitrous Oxide										
		3	5	6	7	10	12	14	15	16	18	20	22	25	30	3	4	5	6	7	8	9	10	11	13	20
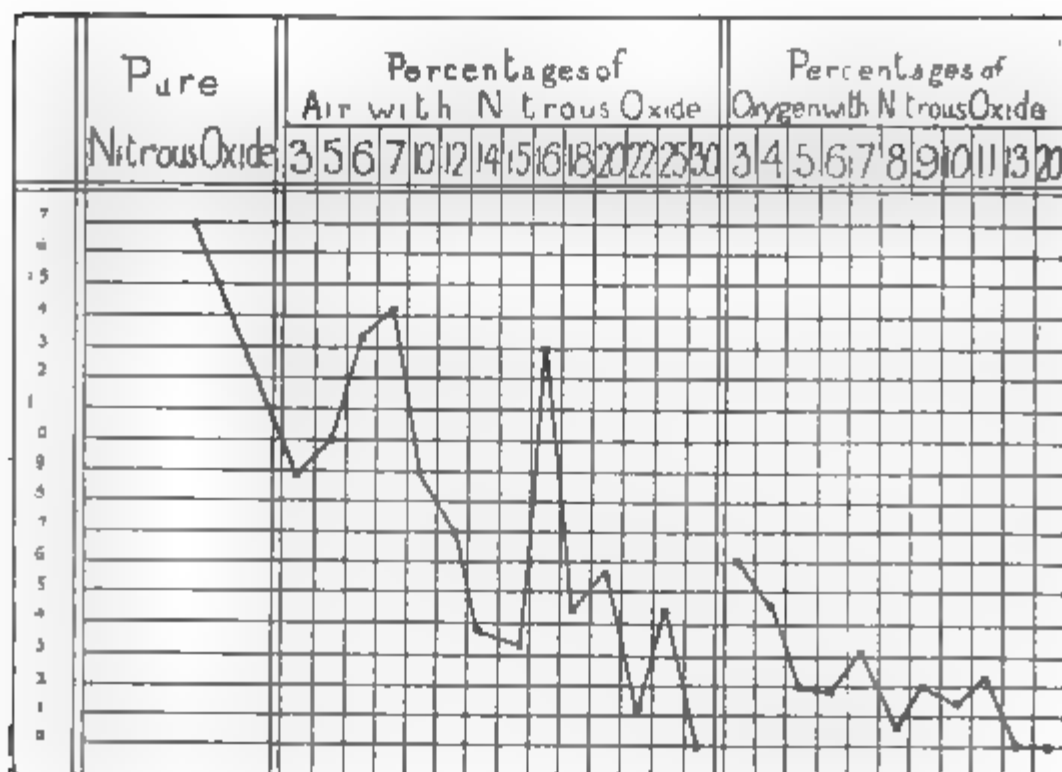
proportion of air increases is well shown. The irregularities observed are doubtless due to the fact that the cases under each percentage were not sufficiently numerous to mask the influences of a few exceptional cases. Thus with 16 per cent. of air there were only five cases, and it is fairly certain that amongst these there must have been one or two patients whose respiratory passages were in such a condition that stertor more readily arose than usual.

Patients with enlarged tonsils, post-nasal adenoid growths, and other conditions capable of narrowing the air-channels, become stertorous more quickly and in a greater degree than those whose air-passages are free. As in other charts, the oxygen cases will be seen to be comparatively free from those irregularities which mark the cases in which air was employed as the oxygenating agent.

In preparing the above chart cognizance was only

taken of stertor which occurred during the administration, or at the moment the face-piece was removed.

Should the inhalation of nitrous oxide be continued beyond the proper limits breathing will cease, either from anoxæmic spasm of respiratory muscles, or from mechanical obstruction at the superior aperture of the larynx. Cessation of breathing from pure and simple paralysis of its nervous mechanism is certainly very uncommon as the result of an overdose of nitrous oxide, and when it does occur is usually due quite as much to cerebral anæmia from a feeble or failing circulation as to the direct effects of the unoxygenated blood upon the respiratory centres.

7. *Phonation during inhalation or first* 30 *secs. after.*—Dealing with the accompanying chart as a whole, it will be seen that the occurrence of phonated sounds during

CHART VII.—*Showing average degree of phonation.*

Arbitrary Numerical Scale.—None = 0; slight = 5; moderate = 15; extreme = 35.
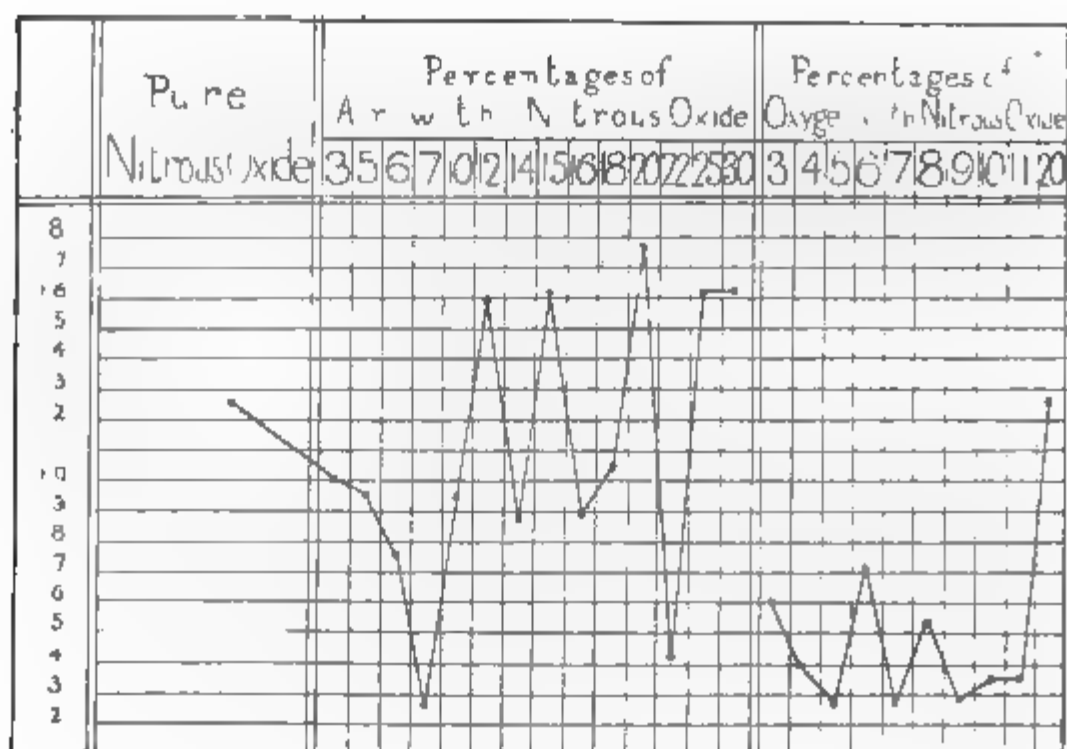
inhalation and the first 30 secs. afterwards was rather more frequent when pure nitrous oxide was given, or when small percentages of air or oxygen were administered

with this gas, than when somewhat greater, though still small percentages of air or oxygen were present in the mixtures. The progressive and steady fall in phonation as we pass from pure nitrous oxide to nitrous oxide with 7 per cent. of air is very obvious. This means that as the percentage of air increased it was found possible to prolong the inhalation, and to obtain a rather deeper anæsthesia, in which phonation was not so liable to arise. The fall in phonation is not so marked in the oxygen cases, although it is present. The next noticeable point in the chart is that in the air cases very considerable phonation was met with in all mixtures containing more than 7 per cent. of air, the most pronounced phonation being recorded in mixtures containing 20 per cent. of air. The air chart is like its predecessors—a very irregular one. The oxygen chart shows far less phonation than the air chart. Indeed, until the percentage of oxygen reached twenty very little phonation was recorded.

8. *Reflex and excitement movements during inhalation or first* 30 *secs. after.*—There is, as the charts show, a very striking difference between the air and the oxygen cases in the matter of reflex and excitement movements. Dealing with the air cases first, it will be seen that, although there are considerable fluctuations, there is a distinct tendency for reflex and excitement movements to lessen as the percentage of air increases. This lessened tendency, however, soon disappears as the percentages continue to increase, and after 14 per cent. has been reached the chart shows a distinct tendency in the opposite direction, the degree of reflex and excitement movement increasing as the percentages of air are also augmented. The maximum reflex and excitement movement was met with when 30 per cent. of air was employed. In the oxygen cases there is, as in the others, an initial but slight tendency for these to decrease as the percentage of oxygen rises, but with this exception the chart shows a gradual and progressive tendency in the other direction, the maximum movements being met with under the

CHART VIII.—*Showing average degree of reflex and excitement movements.*

Arbitrary Numerical Scale.—None = 0; slight = 0; moderate = 20; extreme = 40.

	Pure Nitrous Oxide	Percentages of Air with Nitrous Oxide														Percentages of Oxygen with Nitrous Oxide									
		3	5	6	7	10	12	14	15	16	18	20	22	25	30	3	4	5	6	7	8	9	10	11	20
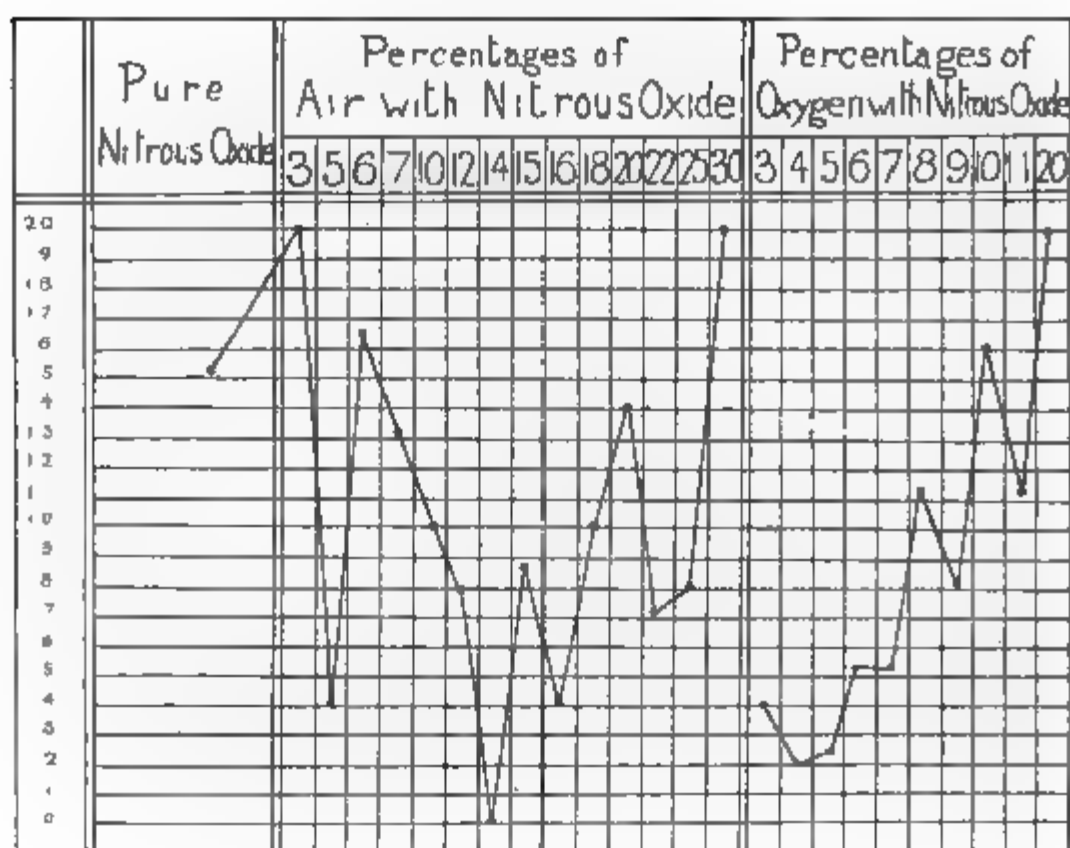
maximum percentage of oxygen (20). The best cases, so far as the absence of inconvenient reflex and excitement movement is concerned were obtained with 14 per cent. of air, and with 4 per cent. and 5 per cent. of oxygen.

9. *After effects.*—So far as unpleasant after effects are concerned, it would appear that these are rather more liable to follow the inhalation of nitrous oxide and oxygen than that of nitrous oxide and air. The difference, however, is but slight.

10. *General result, from the combined points of view of the patient, surgeon, and anæsthetist.*—By the system which has been already referred to it is possible to give an arbitrary number to every case to indicate its value from a broad and general point of view. The accompanying chart will show under what percentages of air and of oxygen the best general results were obtained.

CHART IX.—*Showing the average general result.*

Arbitrary Numerical Scale.—Perfect type = 0; failure to produce
anæsthesia = 200.
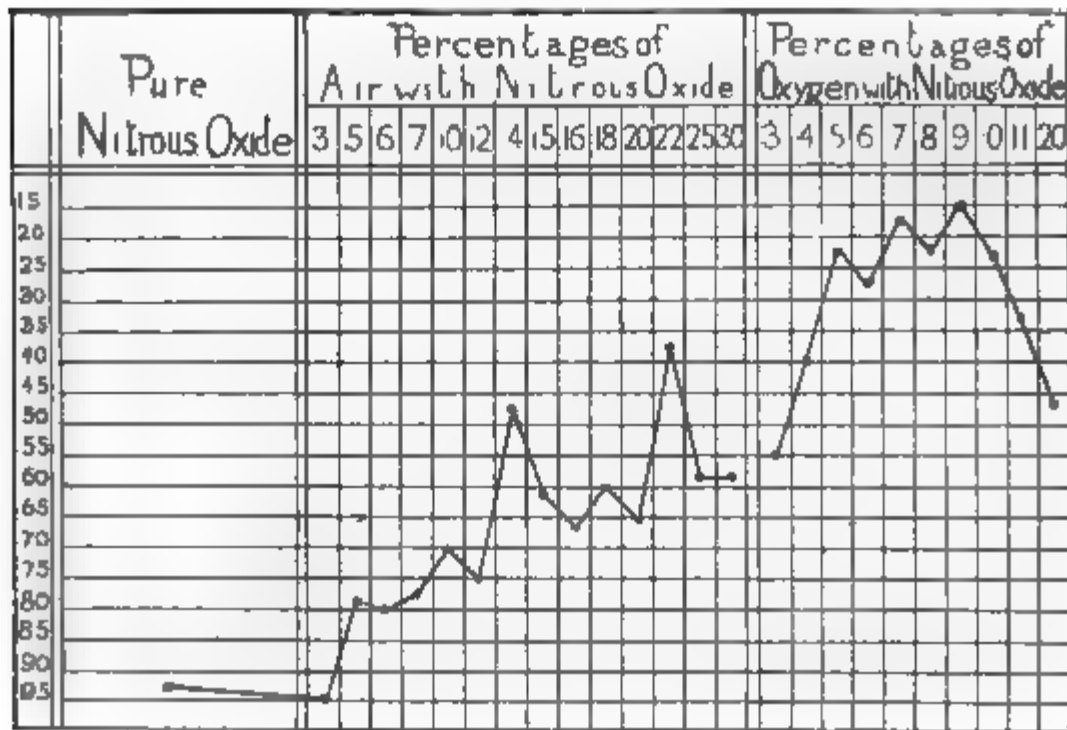
It will be seen that the pure nitrous oxide cases are
very low in the scale; that the best results in the air
cases were obtained with 14 per cent. and 22 per cent.
of air; and that the best results in the oxygen cases were
obtained with 5—9 per cent. of this gas.   The best results
of all are recorded with 9 per cent. of oxygen; but directly
this percentage is exceeded the general results of the cases
begin to decline.   It is interesting to observe a much
higher general value for the cases in which oxygen was
used than for those in which air was employed.

VIII. THE CASES OF SERIES II (NON-COMPARABLE CASES).

Amongst the total of 231 cases dealt with in this
investigation there were five which demand special and
separate consideration; for in all of these there were
conditions present which tended to modify the usual effects
of the gas or mixture administered.   The cases are as
follows.

(1) Case 85 *a. Nitrous oxide free from air or oxygen.*—
Female æt. 43. Sallow complexion; slim build; has pre-systolic mitral murmur. Has been an out-patient at Charing Cross Hospital (see case 81, third case of present series).

Inhalation period.

18 secs., breathing rather restricted. 33 secs., breathing deeper; expiration rather forcible. 51 secs., inspiration irregular. 60 secs., considerable anoxæmic convulsion and stertor, but very little cyanosis. 63 secs., administration discontinued.

Anæsthesia period.

During operation an ashy pallor supervened; aspect alarming; respiration continuing; pulse not observed.

When operation over, pulse very rapid and small, about 160. Two teeth removed. Inhalation 63 secs.; available anæsthesia 24 secs.; 5·02 gallons of nitrous oxide inhaled. " Bad marks " = 150.

(2) Case 93 *a. Nitrous oxide free from air or oxygen.*—
Male æt. 47. Tall; broad shoulders; has thick curly grey beard and moustache.

Inhalation period.

3 secs., good regular breathing. 54 secs., breathing deeper. 75 secs., soft snoring. 87 secs., no conjunctival reflex. 93 secs., colour dusky. 96 secs., slight anoxæmic convulsion. 102 secs., administration discontinued.

Anæsthesia period.

6 secs., no reflex movement or noise. 24 secs., no reflex movement or noise. 36 secs., no conjunctival reflex. 45 secs., anæsthesia at an end.

Five teeth extracted: no knowledge, no pain; very pleasant dream. Inhalation 102 secs. Available anæsthesia 45 secs., 7·4 gallons inhaled. " Bad marks " = 40.

(3) CASE 81. *Nitrous oxide with* 20 *per cent. of air.*— Same patient as in case 85*a* (first case of present series).

Inhalation period.

36 secs., good regular breathing. 48 secs., shuts her eyes when requested. 57 secs., expiration rather forcible. 72 secs., respiration quicker. 75 secs., no conjunctival reflex. 78 secs., mild snoring. 84 secs., quick respiration. 99 secs., colour unchanged. 105 secs., eyes fixed and to the left, pupils medium sized. 120 secs., administration discontinued.

Anæsthesia period.

3 secs., colour slightly dusky. 9 secs., quite quiet. 15 secs., no conjunctival reflex. 18 secs., pupils medium. 27 secs., quite quiet. 33 secs., no conjunctival reflex. 36 secs., very slight phonation; operation over. 45 secs., anæsthesia at an end.

Two teeth removed. Inhalation 120 secs. Available anæsthesia 45 secs. " Bad marks " = 10.

(4) CASE 117 *a. Nitrous oxide with* 10 *per cent. of oxygen.*—Female æt. 31. Same patient as in Case 121 of Series I. Dark complexion, nervous. Corsets not unfastened.

Inhalation period.

3 secs., good regular breathing. 36 secs., holds breath slightly. 57 secs., breathing slightly irregular. 72 secs., breathing very shallow. 90 secs., considerable excitement; has to be held. 111 secs., excitement subsiding. 120 secs., no conjunctival reflex. 123 secs., breathing now regular; excitement subsided. 129 secs., slight snoring. 171 secs., administration discontinued.

Anæsthesia period.

3 secs., no change of colour. 15 secs., breathing quiet. 39 secs., slight phonation. 45 secs., pupils large. 54 secs., considerable phonation. 60 secs., anæsthesia at an end.

Eight teeth removed. No knowledge, no pain; distressing dream; slow recovery. Inhalation 171 secs. Available anæsthesia 60 secs. 9·93 gallons inhaled. " Bad marks " = 85.

(5) CASE 62 *a*. *Nitrous oxide with 5 per cent. of oxygen.*—Male æt. 29. Same patient as in Case 61 of Series I. Healthy-looking; average height; fair moustache; not nervous. Second administration about 10 minutes after first.

Inhalation period.

27 secs., good regular breathing. 42 secs., slight movement of body. 48 secs., deep gasping breathing. 69 secs., considerable movement of body. 75 secs., movement passing off. 78 secs., movement ceased. 81 secs., considerable stertor and rigidity with some duskiness; administration discontinued.

Anæsthesia period.

15 secs., still stertorous. 21 secs., colour normal. 39 secs., anæsthesia at an end.

No pain, no dream. Inhalation 81 secs. Available anæsthesia 39 secs. 10·67 gallons inhaled. " Bad marks " = 100.

An unsatisfactory case, owing to the patient having been anæsthetised but a short time before.

IX. A COMPARISON OF THE EFFECTS PRODUCED IN THE SAME PATIENT BY THE ADMINISTRATION OF DIFFERENT GASES OR GASEOUS MIXTURES.

(1) *Pure nitrous oxide compared with mixtures of nitrous oxide and air.*

CASES 149 (*pure nitrous oxide*) and 164 (*nitrous oxide with 12 per cent. of air*).—Anoxæmic convulsion came on very rapidly in the absence of oxygen, and the face-piece had to be removed after 35 secs. inhalation. With 12 .

per cent. of air the asphyxial "oscillation" was slight, and the inhalation was continued for 42 secs. The available anæsthesia, however, was about the same as with pure nitrous oxide. It is interesting that loud phonation occurred with the 12 per cent. of air during the operation (at 24 secs. in the anæsthesia period), whereas with the pure gas there was no phonation till after 33 secs. The general result of the two cases from the operator's point of view may be regarded as the same. The objectional phenomena numerically expressed were—with pure nitrous oxide 75; with the mixture 75.

CASES 85 *a* (*pure nitrous oxide*) and 81 (*nitrous oxide with* 20 *per cent. of air*) (Cases 1 and 3 in Series II just considered).—The effect of withholding oxygen in the case of a patient with mitral stenosis is very well shown. The state of partial syncope during the operation in case 85 *a* was not noticed in Case 81. The long inhalation (120 secs.) which was rendered possible by the presence of 20 per cent. of air led to a more prolonged and more satisfactory form of anæsthesia (45 as compared to 24 secs.). The phonation in the anæsthesia period of the pure nitrous oxide case also testifies to the less profound anæsthesia. It cannot be regarded as good practice to administer pure nitrous oxide to such a patient as this. The 20 per cent. of air completely prevented asphyxial convulsion, and allowed the pulmonary circulation, already embarrassed by the mitral affection, to proceed in its accustomed manner, and the left heart to receive its usual or almost its usual supply of blood.

(2) *Pure nitrous oxide compared with mixtures of nitrous oxide and oxygen.*

CASES 149 (*pure nitrous oxide*) and 176 (*nitrous oxide with* 8 *per cent. of oxygen*).—The contrast between these cases is very marked. With nitrous oxide free from oxygen there was a rapid onset of asphyxial symptoms in

about 30 secs.; with 8 per cent. of oxygen all obvious asphyxial phenomena were absent, the mixture being a perfectly respirable one. The inhalation with nitrous oxide was 33 secs.; with the mixture 162 secs. The available anæsthsia with the pure gas was 26 secs.; with the mixed gases 42 secs. Although the inhalation lasted so much longer the patient's sensations were of a more pleasant description than when pure nitrous oxide was employed. The objectionable phenomena, numerically expressed, were: with nitrous oxide 75; with the mixture 20.

(3) Mixtures of nitrous oxide and air compared with mixtures of nitrous oxide and oxygen.

CASES 17 (nitrous oxide with 5 per cent. of air) and 32 (nitrous oxide with 5 per cent. of oxygen).—The principal differences between these administrations are (1) the presence of asphyxial symptoms in Case 17, and the absence of such symptoms in Case 32. (2) The shorter inhalation in the air case (63 secs. as compared to 105 secs.) did not lead to a longer anæsthesia (39 secs. as compared to 42 secs.), but to a deeper form of unconsciousness, as is shown by the later onset of reflex movement, and the complete absence of phonation in the oxygen case. The objectionable phenomena, numerically expressed, were, with air 115; with oxygen 15.

CASES 34 (nitrous oxide with 10 per cent. of air) and 39 (nitrous oxide with 10 per cent. of oxygen).—In addition to the presence of anoxæmic symptoms in the air case, and the total absence of such symptoms under nitrous oxide and oxygen, there was a considerable difference in the patient's sensations under the two mixtures, these having been rather unpleasant after the nitrous oxide and air. With 10 per cent. of air the inhalation lasted 75 secs.; the available anæsthesia was 33 secs.; and the objectionable phenomena came to 70. With 10 per cent. of oxygen

the inhalation was 178 secs. ; the anæsthesia was 45 secs. ; and the objectionable phenomena were 0.

CASES 40 (*nitrous oxide with* 12 *per cent. of air*) and 33 (*nitrous oxide with* 5 *per cent. of oxygen*).—A better and deeper form of anæsthesia was secured with 5 per cent. of oxygen than with 12 per cent. of air. Stertor and slight "oscillation" were present on both occasions. With 12 per cent. of air the inhalation lasted 75 secs.; the anæsthesia 30 secs.; and the objectionable phenomena numerically expressed came to 90. With 5 per cent. of oxygen the inhalation was 105 secs., the anæsthesia 40 secs., and the objectionable phenomena amounted only to 45. There was no dream in the oxygen case, and a pleasant dream in the air administration.

CASES 164 (*nitrous oxide with* 12 *per cent. of air*) and 176 (*nitrous oxide with* 8 *per cent. of oxygen*).—The loud phonation during the available anæsthesia period of the nitrous oxide and air inhalation indicated the rather imperfect type of anæsthesia produced by this mixture : at the corresponding point after the nitrous oxide and oxygen administration the patient was perfectly quiet. The inconvenient movement noted in the air case has a similar significance. With 12 per cent. of air the inhalation was 42 secs., the anæsthesia about 26 secs., and the number representing the objectionable phenomena was 75. With 8 per cent. of oxygen the inhalation was 162 secs., the anæsthesia 42 secs., and the number for the objectionable phenomena 20.

CASES 133 (*nitrous oxide with* 15 *per cent. of air*) and 130 (*nitrous oxide with* 5 *per cent. of oxygen*).— Rather forcible expiration was noted with the mixture containing air, but not with that containing oxygen. It is interesting that a state of "physiological apnœa" was recorded with 15 per cent. of air, but not with 5 per cent. of oxygen, the apnœa having come about after the forcible expiration. It is also interesting that the apnœa

came on before consciousness was completely lost, and that breathing recommenced on request about one and a quarter minutes after the inhalation of the nitrous oxide and air had begun. Consciousness was lost almost immediately after this. With nitrous oxide and 5 per cent. of oxygen consciousness was lost about fifteen or eighteen seconds earlier, showing the greater potency of the oxygen mixture, i. e. the greater proportion of the anæsthetic element in it. Slight reflex movement and considerable phonation were noted during the available anæsthesia of the air case, but not during that of the oxygen administration. " Mild oscillation " was recorded during the inhalation of the air mixture, but not during the inhalation of the mixture containing oxygen. With 15 per cent. of air the inhalation lasted 105 secs., the anæsthesia 27 secs., and the objectionable phenomena may be represented by the number 80. With 5 per cent. of oxygen the inhalation lasted 129 secs., the anæsthesia 42 secs., and the objectionable phenomena may be represented by 25.

Cases 168 (*nitrous oxide with* 22 *per cent. of air*) and 187 (*nitrous oxide with* 8 *per cent. of oxygen*).—The tendency towards " physiological apnœa " in Case 187 was observed after the breathing had been rather deeper than usual. In Case 168 there is no record of deep breathing or of apnœa. In the air case the pupils were dilated at 126 secs. of the inhalation period; in the oxygen case they were of moderate size at 126 secs. The numbers are—Case 168, inhalation 135 secs., anæsthesia 42 secs., and objectionable phenomena 5 ; Case 187, inhalation 159 secs., anæsthesia 39 secs.; objectionable phenomena 0.

Cases 195 (*nitrous oxide with* 22 *per cent. of air*) and 184 (*nitrous oxide with* 8 *per cent. of oxygen*).—There was no muscular rigidity during the inhalation of 22 per cent. of air, but rigidity was noted under the 8 per cent. of

oxygen mixture. The "mild oscillation" and "slight cyanosis" met with under the air mixture were not observed with the nitrous oxide and oxygen. There was "loud phonation" at 24 secs. in the anæsthesia period after the air mixture, but no phonation was noted till 54 secs. after the inhalation of the oxygen mixture, and then it was only slight. The numbers are—Case 195, inhalation 117 secs., anæsthesia 27 secs., objectionable phenomena 75; Case 184, inhalation 207 secs., anæsthesia 60 secs., objectionable phenomena 0.

CASES 195 (*nitrous oxide with* 22 *per cent. of air*) and 189 (*nitrous oxide with* 10 *per cent. of oxygen*).—The large percentage of oxygen in Case 189 produced tranquil breathing. The peculiar "secondary rigidity" to which I have elsewhere alluded was met with in this case at 159 secs., and as the conjunctival reflex was present at the time and also afterwards, it would seem that this rigidity is associated with a light rather than with a deep form of anæsthesia. The inconvenient straightening of the body during the operation in Case 189 indicated light anæsthesia, as did subsequent phonation at 39 secs. of the anæsthesia period. It is curious that there was no reflex movement during the operation in Case 195 as one might have expected, seeing that the percentage of nitrous oxide was less in the air than the oxygen mixture. There was, however, loud phonation at 24 secs. of the anæsthesia period in the air case. The numbers are—Case 195, inhalation 117 secs., anæsthesia 27 secs., objectionable phenomena 75; Case 189, inhalation 174 secs., anæsthesia 51 secs., objectionable phenomena 40.

CASES 170 (*nitrous oxide with* 22 *per cent. of air*) and 201 (*nitrous oxide with* 11 *per cent. of oxygen*).—There is very little difference between these two administrations. The anæsthesia seems to have been of a rather better type with 22 per cent. of air than with 11 per cent. of

oxygen. The conjunctival reflex was lost at 132 secs. in the air case, and at 156 secs. in the oxygen case. It is interesting to note the deep and slightly strained expiration even with 11 per cent. of oxygen, and the body movement (rhythmic with expirations) in the same case. With 22 per cent. of air the inhalation lasted 147 secs., the anæsthesia 63 secs., and the objectionable phenomena may be represented by the number 30. With 11 per cent. of oxygen the inhalation lasted 192 secs., the anæsthesia 39 secs., and the objectionable phenomena may be represented by 20.

CASES 127 (*nitrous oxide with* 33⅓ *per cent. of air*) and 142 (*nitrous oxide with* 6 *per cent. of oxygen*).—This comparison is of interest because the percentage of oxygen was practically the same on each occasion. With 33⅓ per cent. of air no true anæsthesia was produced; with 6 per cent. of oxygen a satisfactory result followed. The presence of the large percentage of nitrogen in the former case was doubtless the cause of the failure to produce anæsthesia. Although there is only this one case in which as much as one third of atmospheric air was mixed with nitrous oxide, it would seem that such a mixture is incapable of producing anæsthesia. There are several cases in which 70 per cent. of nitrous oxide with 30 per cent. of air successfully anæsthetised patients.

(4) *Certain mixtures of nitrous oxide and oxygen compared with certain other mixtures of these gases.*

CASES 206 (*nitrous oxide with* 5 *per cent. of oxygen*) and 222 (*nitrous oxide with* 8 *per cent. of oxygen*).—The most interesting point in this comparison is the occurrence of " secondary rigidity " (see p. 171) between 114 and 120 secs. of the inhalation period with 8 per cent. of oxygen, and the absence of this condition with 5 per cent. of oxygen. This is suggestive of the phenomenon which takes place always after anæsthesia has become estab-

lished, being dependent, at all events to some extent, on considerable proportions of oxygen. Why more of the 8 per cent. mixture was used in 120 secs. than of the 5 per cent. mixture in 141 secs. is not clear. The anæsthesia with 8 per cent. of oxygen lasted 33 secs. after 120 secs. inhalation ; with 5 per cent. of oxygen it lasted 37 secs. after 141 secs. inhalation. The objectionable phenomena in the 8 per cent. case are represented by the number 25 ; in the 5 per cent. case by 0.

CASES 206 (*nitrous oxide with* 5 *per cent. of oxygen*) and 200 (*nitrous oxide with* 11 *per cent. of oxygen*).—There is very little difference between these two administrations. There was rather more reflex movement during the operation in the 11 per cent. of oxygen case than in the other. In both cases an extremely satisfactory state was obtained. The patient was a little lachrymose after the higher percentage of oxygen.

CASES 184 (*nitrous oxide with* 8 *per cent. of oxygen*) and 189 (*nitrous oxide with* 10 *per cent. of oxygen*).—" Secondary movement " was observed after 159 secs. inhalation of 10 per cent. of oxygen, but no such phenomenon occurred with 8 per cent. of oxygen. With the larger percentage of oxygen reflex straightening of the body during the operation was met with, whereas with the smaller percentage there was no reflex at all. It must be remembered, however, in making these comparisons that the 8 per cent. of oxygen mixture was administered for 207 secs., whereas the 10 per cent. mixture was given only for 174 secs. It is interesting to note that 33 secs. after the rather protracted administration of the 8 per cent. mixture the patient was quite relaxed. The available anæsthesia was, after 207 secs. inhalation of the 8 per cent. mixture, 60 secs.; after 174 secs. inhalation of a 10 per cent. mixture, 51 secs. The objectionable phenomena may, in the 8 per cent. case, be represented by 0; in the 10 per cent. case by 40. The

comparison of these two administrations only strengthens the opinion formed by comparing other cases, viz. that 10 per cent. of oxygen is rather too high a percentage if we wish to obtain the best type of nitrous oxide anæsthesia.

CASES 222 (*nitrous oxide with* 8 *per cent. of oxygen*) and 200 (*nitrous oxide with* 11 *per cent. of oxygen*).—With 8 per cent. of oxygen the conjunctival reflex disappeared at 78 secs. ; with 11 per cent. of oxygen there was very slight conjunctival reflex at 99 secs. "Secondary movement" was met with late in the administration of the 8 per cent. mixture, but not with the 11 per cent. mixture. Although the inhalation of the 11 per cent. mixture was carried on for 147 secs., the resulting anæsthesia was not of such a satisfactory type, *i. e.* not so deep as that produced by the inhalation for 120 secs. of an 8 per cent. mixture. This is shown by the reflex movement early in the operation in the former case, and the absence of such movement in the latter. It is interesting that, although the inhalation lasted longer in the 11 per cent. case, the quantity of the mixture used was smaller. This is accounted for by the sleep-like state with quiet breathing which was brought about in the 11 per cent. case. The smaller proportion of oxygen in the other case led to deeper breathing, and hence to the inhalation of more of the mixed gases. The available anæsthesia was the same in both cases. The objectionable phenomena numerically expressed were in each case 25.

CASES 117 *a* (*corsets tight*) and 121 (*corsets loose*).—A 10 per cent. mixture of oxygen and nitrous oxide was used on each occasion. In Case 117 *a* the restricted breathing culminated in excitement which necessitated the patient being held ; in Case 121 no such excitement was observed. The most probable explanation for the occurrence of the excitement is that the residual air originally

present in the bases of the lungs took a comparatively long time to be ousted by the mixture, so that a comparatively long time was needed for the nitrous oxide to become absorbed by the blood in sufficient quantities to produce anæsthesia. Slow absorption seems to favour the development of these intoxication effects. A distressing dream was experienced on the occasion when the corsets were not unfastened, but nothing of the kind was complained of on the subsequent occasion. When the corsets were loose there were no objectionable phenomena, but when they were not unfastened objectionable symptoms occurred to an extent which may be expressed by the number 85.

X. Conclusions.

From the foregoing investigation the following conclusions may be deduced.

1. When pure nitrous oxide is administered to the human subject in such a manner that no free oxygen gains admission during the administration, certain phenomena arise, which for our present purposes may be regarded as being either—

(a) Phenomena of anæsthesia, or

(b) Phenomena of asphyxia.

2. The anæsthetic phenomena of nitrous oxide, although apparently very different from those of ether or chloroform, are in their essential features remarkably similar.

3. The most conspicuous of the asphyxial phenomena of pure nitrous oxide are—

(i) Embarrassed and deeply stertorous breathing;

(ii) Cyanosis; and

(iii) Anoxæmic convulsion.

All these may be eliminated, without interfering with the anæsthetic effects of the gas, by administering with it certain proportions of oxygen, either pure or as atmospheric air.

4. There are other less obvious asphyxial phenomena,.

such as wide dilatation of the pupils, swelling of the tongue and adjacent structures, and rapid cardiac action, which, like the more important symptoms above referred to, may be prevented or modified by similar means.

5. Under the influence of pure nitrous oxide, breathing becomes deeper and quicker than usual. At the end of from 55 to 60 secs. its rhythm becomes altered either by—

(a) Obstructive stertor;

(β) Anoxæmic convulsion attacking the respiratory muscles; or

(γ) Both conditions combined.

If the administration be pushed far enough, breathing becomes arrested in one of these ways. Paralytic cessation of breathing is very rare, and when it occurs is dependent quite as much upon cerebral anæmia from defective circulation as upon the presence of un-oxygenated blood in the vessels supplying the respiratory centres.

6. The deep and obstructive stertor of pure nitrous oxide narcosis is not met with when employing mixtures containing moderate percentages of air or oxygen. With such mixtures only softly snoring breathing is produced. When the percentage of air or oxygen is considerable (30 per cent. of air or 13 per cent. of oxygen), respiration becomes noiseless and free from all obstruction.

7. The most marked cyanosis is met with when very small percentages of air (3 to 6 per cent.) or oxygen (under 3 per cent.) are administered with nitrous oxide (see p. 198). With pure nitrous oxide cyanosis may not have time, as it were, to become pronounced, for the administration may be cut short by deep stertor. As the percentage of air or oxygen increases, cyanosis lessens, till with 30 per cent. of air it is very slight, and with 11 per cent. of oxygen it disappears altogether.

8. Anoxæmic convulsion, like cyanosis, is liable to be greater with small percentages of air or oxygen than with pure nitrous oxide itself, and for the reason just given.

But as the percentages of air or oxygen increase the convulsion decreases, till with 30 per cent. of air and 6 per cent. of oxygen respectively it ceases to occur.

9. Reflex and excitement movements are most likely to arise either with pure nitrous oxide, or with nitrous oxide mixed with small percentages of air (3 to 7 per cent.), or with nitrous oxide mixed with considerable percentages of air (20 to 30 per cent.) or oxygen (10 to 20 per cent.). They are least likely to assert themselves with mixtures containing moderate quantities (12 to 16 per cent.) of air, or moderate quantities (3 to 7 per cent.) of oxygen.

10. Phonated sounds are most common when nitrous oxide is administered with large percentages of air (12 to 30 per cent.) or oxygen (20 per cent.). When nitrous oxide is administered pure, or with small percentages of air (3 to 5 per cent.), they are met with to a moderate extent only. Phonation is least likely to arise with nitrous oxide and air mixtures when air is present to the extent of from 6 to 10 per cent., and with nitrous oxide and oxygen mixtures when oxygen is present to the extent of from 3 to 11 per cent.

11. The duration of available anæsthesia after inhalation was found to be longest after the administration of mixtures containing from 3 to 11 per cent. of oxygen, the maximum duration having been attained with 7 per cent. mixtures. With nitrous oxide and air mixtures the resulting anæsthesia was distinctly longer than with pure nitrous oxide, but the results were very uncertain in these cases. The shortest available anæsthesia was recorded with nitrous oxide alone, and with nitrous oxide mixtures containing 30 per cent. of air.

12. In estimating the respective merits of the various mixtures used as anæsthetic agents, the investigation shows that the best results were obtained with mixtures of nitrous oxide and oxygen ; the next best with mixtures of nitrous oxide and air ; and the worst with nitrous oxide free from air or oxygen. Air is not nearly so

suitable as oxygen for eliminating the asphyxial factors of a pure nitrous oxide administration, owing to the large volume of useless nitrogen which must necessarily be inhaled. The nitrous oxide and air cases were, as compared with those of nitrous oxide and oxygen, far more uncertain in their course.

13. There is no one mixture of nitrous oxide with air or with oxygen which will successfully anæsthetise every patient.

14. In order to obtain the best form of anæsthesia, oxygen should be administered with nitrous oxide by means of a regulating apparatus, the percentage of the former gas being progressively increased from 2 per cent. or 3 per cent. at the commencement of the administration, to 7, 8, 9, or 10 per cent. according to the circumstances of the case. The longer the administration lasts, the greater may be the percentages of oxygen admitted.

15. The next best results to those obtainable by means of a regulating apparatus for nitrous oxide and oxygen are to be secured by administering certain constant mixtures of these two gases. Mixtures containing 5, 6, or 7 per cent. of oxygen are the best for adult males, and mixtures containing 7, 8, or 9 per cent. are the best for females and children.

16. The next best results to those last mentioned are to be obtained by means of mixtures of nitrous oxide and air, from 14 to 18 per cent. of the latter being advisable in anæsthetising men, and from 18 to 22 per cent. in anæsthetising women and children.

17. Paul Bert, whose valuable researches upon the effects produced by the inhalation of nitrous oxide and oxygen are well known, regarded an increased atmospheric pressure as essential to the production of anæsthesia by means of these gases. It is clear, however, from the foregoing investigation, that deep and satisfactory anæsthesia, unaccompanied by any obvious asphyxial manifestations, may be secured, at ordinary pressures, by mixtures of nitrous oxide and oxygen containing even

as much of the latter gas as is present in our atmosphere.

18. The last-mentioned fact would seem, at first sight, to give a death-blow to the asphyxial theory of nitrous oxide anæsthesia—a theory which supposes that this gas produces its anæsthesia by limiting or arresting normal oxidation processes within the central nervous system. But are we justified in assuming that because no *obvious* asphyxial manifestations make their appearance during the inhalation, no such interference with oxidation is taking place ?

(For report of the discussion on this paper, see ' Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. xi, p. 55.)

A CASE

OF

EXTIRPATION OF A LARGE NON-PULSATING ANEURYSM

INVOLVING THE COMMON, INTERNAL, AND EXTERNAL CAROTID ARTERIES OF THE RIGHT SIDE OF THE NECK

WITH

REMARKS ON NON-PULSATION IN ANEURYSMS AND THE TREATMENT OF ANEURYSMS BY EXTIRPATION

BY

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I MAY say at once that neither by myself nor my colleagues was the swelling in this patient's neck diagnosed as an aneurysm. Indeed, it was only at the operation undertaken for the purpose of exploring and, if possible, of removing what was believed to be probably a malignant tumour, that the true nature of the swelling

was discovered. There was absolutely no pulsation to be felt, and although there was some difference of opinion as to whether the swelling was malignant or innocent, all who saw it considered that it was a solid growth, probably of glandular origin.

A. B—, a ship's steward, 49 years of age, was sent to me by my friend Mr. W. H. Johnson, under whose care he had been for some time. He stated that some six years ago he first noticed a little lump in the right side of the neck, which had grown slowly and without pain until four months ago, since which time it had begun to increase rapidly. At first it was hard, painless, not tender on pressure, and felt to Mr. Johnson like an enlarged gland. Some of the teeth on the right side were discovered to be carious, and two were extracted, but without influencing the swelling.

The patient, a stoutish, florid, short-necked, and healthy-looking man, had hitherto enjoyed excellent health, and his family history was good. There existed on the right side of the neck, extending from below the right ear and angle of the jaw almost down to the clavicle, a swelling the size of a cricket ball. It reached nearly to the middle line in front, and behind passed deeply beneath the sterno-mastoid muscle. It was roughly globular in shape with a somewhat irregular surface, and was gene-rally hard in consistency, but soft and semi-fluctuating in places. The skin over it moved freely, and although it clearly had deep connections it was not fixed to bone, nor to the larynx, which moved independently both verti-cally and laterally. No pulsation was present, and nothing in the form of a primary growth was discoverable either in the mouth, pharynx, or larynx. Breathing and swallowing were quite natural; the pupils were equal; and nothing was noticed abnormal in the pulse; in short, the only trouble complained of was the inconvenience in moving the neck, due to the large size of the swelling, and a shooting pain, like earache, in the right ear at night-time. A medium-sized exploring syringe was passed

deeply into the tumour at one of the soft spots, but only a little glairy fluid was obtained, the piston being sucked back when released from pressure. I came to the conclusion that the swelling was primarily glandular, and that its recent increase made it probable that it was now malignant in nature. The patient was placed on medium doses of liquor arsenicalis, and in November, 1895, he was shown at our weekly consultations, when those of my colleagues who were present considered the tumour was now probably malignant. There appeared some doubt, however, whether it might not be merely inflammatory, and the majority advised that an exploratory incision should be made. All agreed that if the growth were found malignant the incision should be closed, as it was felt, considering the size of the growth and its evident deep connections, that its extirpation under these circumstances could only be accomplished with extreme difficulty and risk.

The state of the case having been fully placed before the patient, he decided, after consultation with Mr. Johnson, to have an exploratory incision made. On December 5th, 1895, with the assistance of Mr. Berry, our then surgical registrar, I proceeded to explore the growth. Having placed the patient under chloroform I made an incision about three inches in length over the tumour along the anterior border of the sterno-mastoid. The tumour when first exposed appeared to be well encapsuled though slightly adherent to the neighbouring parts. When cut into it was of a greyish colour and firm consistency. Seeing that the growth was well encapsuled and was now obviously not malignant, I determined, with the concurrence of Mr. Berry, to attempt its removal. Mr. Berry at this stage of the operation was of the opinion that we were probably dealing with an old thyroid cyst. Whilst, however, cautiously continuing the enucleation, a sudden gush of darkish blood took place from the exploratory incision previously made in the tumour, but was controlled by the fingers, sponges,

and gauze. For the first time it dawned upon us that we were dealing with an aneurysm ; we felt, however, at this stage of the proceedings there was nothing left but to complete its extirpation.

Accordingly, whilst Mr. Berry controlled the hæmorrhage, I cautiously dissected downwards towards the clavicle till the common carotid was discovered entering the lower and outer part of the sac. Two stout silk ligatures were applied, and the artery divided between them. The tumour was now freed from its deep connections, special care having to be taken in dissecting off the vagus nerve, which ran along and was adherent to the outer wall of the sac. The internal jugular vein was not seen ; it was certainly not adherent to the tumour, and was not tied. It may have been obliterated by the pressure of the aneurysm. The dissection was next continued around the upper part of the tumour, and this was the most difficult step in the operation, since the tissues were a good deal matted together, and the aneurysm ran very deeply under the jaw and upwards towards the parotid gland. Finally, the dissection left the aneurysm at its upper part attached by only a pedicle about as large as a little finger, which was evidently the dilated internal carotid artery, the main external carotid artery having probably been tied amongst the number of large vessels which had to be secured during the dissection at the upper, anterior, and internal part of the tumour. After ligature of the pedicle the aneurysm was cut away, a very small piece of the sac being left with the stump. The operation lasted about an hour. Towards the end the patient got rather faint, especially when the vagus nerve was being handled, but the loss of blood was not great. The aneurysm wall was well marked everywhere. It was thinnest at the inner and back part close to the pharynx. At the anterior part the clot was very dense and was decolourised, but at the back and inner part it was dark and recent. It was evident, therefore, that the aneurysm was extending backwards and inwards, and that

before long it would probably have ruptured into the pharynx or cellular tissue of the neck. The huge wound, having been thoroughly irrigated and cleansed with perchloride of mercury solution (1 in 2000), was closed, except at its lowest part, where a drain-tube was inserted. A voluminous pad of sal-alembroth gauze and wool was firmly bandaged on, so as to ensure as much as possible the tissues being in contact. The subsequent history of the case was uneventful. The temperature, with the exception of a rise to 100° on the second day, remained subnormal throughout. The wound healed by the first intention save at the situation of the drain-tube and at one other spot a little above, which subsequently opened, and from which some of the deep ligatures continued to be discharged for some weeks. The patient was kept for the first fortnight with his head fixed between sandbags on moderately low diet, so as to reduce arterial tension as much as possible. He was discharged from the hospital on January 19th, 1896.

Remarks.—I was fortunate in having asked my colleagues to see this case with me in consultation, or I might have laid myself open to the charge of having carelessly overlooked an aneurysm. The fact that the tumour might be an aneurysm had not escaped me. Before puncturing with the exploring syringe I carefully examined for pulsation, and noted at the time in my casebook that there was none present. My surgical colleagues who saw the case with me, Mr. Berry, and the house surgeons, can also vouch for the absence of pulsation. Even when the sac was fully exposed in the exploratory incision and during its enucleation no pulsation was detected. The absence of pulsation, I take it, was due to the aneurysm having become almost consolidated except at its upper and back part, where it was encroaching on the pharynx, and it seems possible that had a finger been passed down that cavity pulsation might have been felt through the pharyngeal wall. The mouth, pharynx, and larynx were explored with the

laryngoscope for the purpose of excluding a primary growth in these situations, but as nothing was seen and the patient had no difficulty in swallowing, a digital examination was not made. The non-escape of blood on puncture, I presume, was due to the needle having entered the consolidated portion. Non-pulsation in an aneurysm, except when it has become consolidated, diffused, or inflammation or suppuration has occurred in the sac, is apparently a somewhat rare phenomenon, for my friend Mr. Miles has made a search for such in medical literature, and the only recorded instances he has been able to find are those mentioned by Mr. Holmes in his 'System of Surgery,' those related by the late Mr. Morrant Baker in the fifteenth volume of 'St. Bartholomew's Hospital Reports,' a case recorded by Dr. Karewski in the 'Berliner klinische Wochenschrift,' and a case of Dr. Schopf in the 'Wiener klinische Wochenschrift.' " Sometimes," says Mr. Holmes, " after an aneurysm has ceased to pulsate and become stationary, it will again commence to grow, and the pulsation will recur. In a case of this kind which was furnished by a preparation to the museum of St. George's Hospital, an increase in size was noticed without any return of pulsation. The artery had been tied above the tumour, and the increase of the latter, which was very marked, from the size of a hen's egg to that of an ostrich's in twelve months, led to the suspicion that the diagnosis had been mistaken, aad the disease not aneurysmal but malignant. After death the accuracy of the original diagnosis was established, but the cause of the absence of pulsation remained obscure."

Mr. Holmes also quotes a case of a woman under Mr. Vincent's care at St. Bartholomew's Hospital, in which the absence of pulsation was attributed to the orifice being at the deep surface of the tumour, and the force of the stream probably broken by clots. The diagnosis of aneurysm in this case was at length verified by an exploratory incision, and amputation was performed.

Mr. Morrant Baker, in his instructive paper in the 'St. Bartholomew's Hospital Reports,' relates two cases under his own care in which, on account of the absence or feebleness of pulsation, much doubt existed as to whether the tumour was an aneurysm or a malignant growth, and one under the care of Mr. Hulke in which like difficulty existed. He also refers to three other cases, two known to Mr. Maunder, and one to a friend whose name is not given. The sequel showed that these cases were aneurysmal. Mr. Baker believed that the absence of pulsation in them was due to leakage and the consequent effusion of blood into the tissues. In the three cases that came under his own cognizance this explanation seems probably correct, but it hardly applies to my case, in that the wall of the sac was everywhere well marked though thinned posteriorly.

The case related by Dr. Karewski in many respects resembles my own. The aneurysm was of large size, reaching from the jaw almost to the clavicle; it involved the carotid arteries, and there was absolutely no pulsation nor bruit. During the operation no pulsation was felt, and on puncture with an exploring trocar blood flowed in a black non-pulsating stream. It was thought to be either a growth or a blood-cyst in connection with a vein, and its aneurysmal nature was only discovered, as in my case, during the operation. The carotids were tied, the clots turned out, and the wound plugged, the patient making a good recovery. Dr. Karewski offers no satisfactory explanation of the absence of pulsation. In Dr. Schopf's case there was no pulsation, neither could any history of such be obtained. It was consequently diagnosed as a growth, and only discovered to be an aneurysm at the operation undertaken for its removal. The absence of pulsation may be attributed to the thickness of the wall of the sac and the presence of firm blood-clot in the interior. The aneurysm seems to have been undergoing a process of consolidation.

The treatment of spontaneous aneurysm by extirpation

has received some attention during the last few years, and several cases have been recorded in which this method has been successfully employed in the treatment of spontaneous aneurysms in the extremities, but I have only been able to discover two cases in addition to my own in which a spontaneous aneurysm of the carotid has been dealt with in this way, namely, the case of Dr. Karewski already referred to, and that of Mr. Henry Morris in the sixty-fourth volume of our 'Transactions.' In Mr. Morris's case a return of pulsation and increase in size had occurred after ligature of the common carotid, and the old operation of laying open the sac, turning out the clots, and securing the vessels feeding the aneurysm was successfully performed. Mr. Morris in his paper refers to the well-known cases of Mr. Syme and Mr. Frothingham, in which traumatic aneurysms of the common carotid were successfully incised, and Mr. Briggs in the 'Journal of the American Medical Association' for 1886, and Herr Saltzmann in the 'Finska läkaresällsk förh.,' vol. xxiv, relate somewhat similar cases.

In the cases of Karewski, Morris, Syme, Frothingham, Briggs, and Saltzmann, however, the old operation of laying open the sac and turning out the clots was employed; but in a case of *traumatic* aneurysm recorded by M. Delargnière the tumour was dissected out after the common carotid had been divided between two ligatures as in my own case. In my own and in Karewski's case it was not known previous to the operation that we were dealing with an aneurysm. In Delargnière's case the dissecting out appears to have been undertaken deliberately. Had the aneurysmal nature of the swelling been recognised in my own case I do not think it would have been possible to apply a ligature to the common carotid for want of available space between the tumour and the clavicle; and had it been possible I doubt if it would have been successful, since, as in Mr. Morris's case, so many large vessels opened into the upper part of the sac. At the same time I do not know that I should have

cared to undertake what would have appeared to me with my then experience the very formidable operation of dissecting out the sac. But from the comparative ease with which the enucleation was performed, the only trouble we had being that from the escape of blood through the exploratory incision in the sac, I would submit that in the case of large aneurysms in this situation in which the common carotid cannot be ligatured, and in which the aneurysm involves several large vessels as here, extirpation holds out the best chances of success ; and I am not sure that even in the case of small carotid aneurisms it would not be safer to dissect out the sac than to ligature the carotid, since by so doing the risk of setting free emboli, as pointed out by M. Delargnière, would be probably much diminished. Had there been no hæmorrhage from the sac to control, nothing would have been easier than the enucleation, for after the proximal artery had been divided between the two ligatures the sac was readily dissected up from the neighbouring structures, the large vessels communicating with its upper and inner part were secured without any particular difficulty, and by drawing the sac down from beneath the angle of the jaw the main internal carotid was brought well into view. The only real trouble lay in controlling the bleeding from the wound in the sac during the necessary movements involved in dissecting it out, and thus keeping the field of operation clear of blood.

A consideration of my case led me to review the wider question of extirpation of aneurysms in general, so with the help of my friend Mr. Ernest Miles I looked up the cases of aneurysm treated by extirpation that have been published during recent years. A large number have been recorded. The majority of these, however, are of traumatic origin, and many of quite small size and situated on medium-sized vessels, as the radial or tibials. Since such aneurysms present no particular interest I have not tabulated them. I have myself dissected out

five or six small traumatic aneurysms during the last year
or so, and I imagine that most surgeons have had a
similar experience.

Dismissing, then, the traumatic aneurysms, I have col-
lected and tabulated thirty-three cases of large idiopathic
aneurysms treated by extirpation. I know of others, the
notes of which, however, are too brief to be of use, and I
have been unable to gain access to the periodicals, &c., in
which an original account of them is published.

Of the thirty-three cases of spontaneous or pathological
aneurisms here tabulated all except two were apparently
the result of atheromatous changes in the artery in which
they were situated. Seventeen involved the popliteal,
six the common or superficial femoral, three the common
carotid, two the junction of the external iliac and femoral,
two the subclavian and axillary, one the subclavian, one
the upper part of the posterior tibial, one the third per-
forating branch of the profunda. The operation of ex-
tirpation was undertaken in ten cases on account of other
surgical methods, such as compression, flexion, or ligature,
having previously failed; in four on account of the aneu-
rysm having become diffused; in four because rupture
of the sac seemed imminent; and in six because the
aneurysmal nature was not recognised, and the swelling
was thought to be a new growth. In nine the reason for
the choice of operation is not stated.

In five cases, including my own, the aneurysm was
removed by first tying the artery on the proximal side in
two places, dividing the artery between the two ligatures,
dissecting out the sac, and removing it after the artery
or arteries had been ligatured on the distal side.

In fifteen the sac was exposed, the artery ligatured on
the proximal and distal side of the aneurysm, and the sac
then removed entire.

In three cases leaking had occurred and a false sac
formed. This was laid open, the clots were turned out,
the artery was then tied at the proximal and distal sides
of the true sac, and the latter removed entire.

In three cases after the sac had been exposed and the artery tied on the proximal and distal side, the sac was opened, the clots turned out, and the sac plugged. In seven cases the sac was opened, the clots turned out, and the artery then secured on the proximal and distal side. In four of these the sac was not removed; in the other three it was removed entire or piecemeal.

Thus three chief methods of extirpation were employed :

1. The old operation of laying open the sac, turning out the clots, and ligaturing the artery above and below.

2. The same operation *plus* the removal of the sac by dissection.

3. Removal of the sac after the artery had first been tied above and below.

The last method, slightly modified as in my case and in four others, I would submit is the ideal way of extirpating an aneurysm. For after two ligatures have been applied to the artery on the proximal side, and the artery has been divided between them, the aneurysm, which remains full of blood and retains its form, can be more readily dissected up from its deep connections than when the sac has been previously opened, and each step of the dissection can be clearly seen, since the ligature on the stump of the vessel entering the sac prevents the sac collapsing. By working round the sac from the proximal towards the distal end, the distal artery is more readily discovered than it is by searching for it at the distal part of the sac, and with less disturbance of the parts; and any large secondary branches that may enter the sac are easily found in the course of the dissection, and can be promptly secured.

All the cases recovered except three, and in one of these fatal cases death was due to erysipelas and suppuration of the knee-joint, the result of the wound becoming septic; in another to gangrene of the limb, amputation being refused; and in another to erysipelas of the wound and the formation of a second aneurysm at

the seat of proximal ligature. In the first of these cases the wound was left open and plugged, and the patient was an alcoholic. The treatment of the wound in the second case is not stated, but the vein was wounded and tied. In the third case the wound became infected with erysipelas, and the patient died one hour after ligature of the innominate and carotid, which was undertaken to arrest the bleeding from the second aneurysm, fifty-nine days after the operation of extirpation.

Gangrene occurred in only three cases (and in one of these was limited to a small patch on the sole of the foot) although in twelve of the cases a portion of the vein corresponding to the aneurysm was extirpated or tied. There does not appear, therefore, to be more risk of gangrene after extirpation than there does after the Hunterian ligature; indeed, it is possible there is less, since the extirpation of the tumour is likely to relieve pressure on the collateral vessels. There is, moreover, less danger of emboli, and in that the main artery is obliterated at only one spot instead of at two, there is better chance of an efficient collateral circulation becoming established.

In thirteen cases the wound healed by first intention. In twelve cases a plug was employed and the wound healed by granulation. In one secondary hæmorrhage occurred. In this case the sac was laid open and not removed. In one a second aneurysm formed above the proximal ligature.

The aneurysms for which extirpation would appear most suitable are these :

1. Where there is insufficient room to apply a ligature to the artery on the proximal side, or where a proximal ligature is attended with great risk, as ligature of the innominate for subclavian aneurysm.

2. Where a number of large vessels communicate with the sac.

3. Where other methods have failed to cure the aneurysm.

4. Where the aneurysm, as in the popliteal artery,

has become diffused, or rupture of the sac or gangrene of the limb is threatened.

5. Where the setting free of emboli, as in carotid aneurysms, would be attended with risk of cerebral softening.

Lastly, the extirpation of an aneurysm in a suitable case, *versus* ligature of the proximal or distal artery, holds out the further advantage that, inasmuch as the whole aneurysm is got rid of, the cure is radical, and all risk of recurrent pulsation, passive enlargement without pulsation, secondary hæmorrhage and inflammation and suppuration of the sac, and later, cicatricial contraction with consequent involvement of the nerve trunks and the impairment of the mobility of joints is removed. From a review of the cases, and from the experience gained in my own, I think it is shown that the operation of extirpation, although no doubt a more formidable one than simple proximal ligature, may, if all modern surgical precautions are taken, be looked upon as a most successful method of treating an external aneurysm, and is perhaps the best that can be adopted for the conditions that have been mentioned. Such a review, however, hardly supports the contention of those surgeons who advccate that the excision of an aneurysm should be the routine treatment. For an ordinary popliteal aneurysm without complications, enucleation can hardly be compared with such a simple and, as a rule, satisfactory procedure as ligature of the popliteal, or even of the femoral, at the apex of Scarpa's triangle. Thus on referring to the tables it is seen that though in some of the cases the enucleation was accomplished readily, in others there was much difficulty in freeing the sac from the external and internal popliteal nerves and the popliteal vein. Indeed, in several cases the vein was injured, or was so adherent that some inches of it had to be removed, whilst in other cases the sac walls were found so thin that the enucleation could not be accomplished, and the sac had to be laid open, with the attendant difficulties. In one case the operation is said to have

taken three hours. In one the sac ruptured during the dissection, and alarming hæmorrhage occurred. Between ligature of the proximal vessel, when this is applicable, and the old operation of extirpation by opening the sac, turning out the clots, and seeking for the artery above and below, at any rate, there can hardly be any comparison.

A table is appended of the thirty-two cases of spontaneous aneurysms treated by extirpation; and a second table of nine cases of carotid aneurysm, both spontaneous and traumatic, treated in like manner. All these nine cases recovered. In two the sac was dissected out after the artery had been tied above and below. In the remaining seven an incision was made into the aneurysm, the clots turned out, and the vessel then secured. Three were spontaneous aneurysms, and are already quoted in the first table; the other six were traumatic.

TABLE OF CASES.

Nine Cases of Carotid Aneurys[

No.	Reference.	Surgeon.	Sex.	Age.	Cause.	Artery affected.	Other treatmen[adopted.
1	Unpublished, 1895	Walsham	M.	49	Spontaneous	Common carotid	None
2	Arch. Prov. de Chir., p. 225, 1896	Delar-genière	F.	16	Traumatic	Common carotid	Nono
3	Observations in Clinical Surgery	Syme	—	—	Traumatic	Common carotid	None; recent case
4	Amer. Journ. Med. Sciences, vol. ii, p. 433	Frothing-ham	—	23	Traumatic	—	Recent case
5	Journ. of Amer. Assoc., p. 553, vol. vi, 1886	Briggs	M.	Young	Traumatic	Common carotid	None; five weeks' duration
6	Med.-Chir. Trans., vol. lxiv, 1881	Morris	F.	45	Spontaneous	External carotid	Common carotid tied
7	Finska Läkare-sällsk förh., xxiv, p. 56, 1885	Saltzmann	—	—	Traumatic	Carotid	Not stated
8	Berl. klin. Woch., 1891, S. 909	Karewski	—	—	Spontaneous	Common carotid	—
9	Brit. Med. Journ., vol. i, 1898, p. 517	Annandale	—	—	Traumatic	Common carotid	None; not room to secur[carotid below sac

Twenty Cases of Spontaneous Aneurysm in which Sac was expose[

No.	Reference.	Surgeon.	Name.	Sex.	Age.	Locality.	Artery affected.	Other treatmen[adopted.
1	Frenken, Über Aneurysme und die Chirurg., Inaug. Diss., Bonn, 1886	Trendelen-burg	—	M.	40	Thigh	Femoral	Digital compression

treated by Extirpation.

Symptoms.	Operation.	Result.
Large non-pulsating swelling; no signs of aneurysm	Sac dissected out after common carotid divided between two ligatures	Recovery.
Well-marked sac size of orange	Sac dissected out after common carotid divided between two ligatures	Recovery.
Large extravasation into neck	Incision; clots turned out; artery ligatured	Recovery.
Large extravasation into tissues of neck becoming circumscribed and pulsating	Incision; finger passed into sac to control bleeding; artery and vein tied above and below wound	Recovery.
Pulsating swelling in neck	Incision; sac opened; artery tied above and below	Recovery.
Recurrent pulsation and increase in size	Incision; clots turned out; arteries tied above	Recovery.
Not mentioned	Incision	Recovery.
Thought to be a tumour or cyst; no pulsation; no bruit	Incision; clots turned out; artery ligatured above and below	Recovery.
Diffuse aneurysm following ligature of common carotid for sarcoma of face	Incision; clots turned out; artery tied above and below	Recovery.

Artery tied on Proximal and Distal Side, and Sac removed.

Symptoms.	Operation.	Result.	Remarks.
Pulsating swelling, size of hen's egg; usual signs of aneurysm	Longitudinal incision over swelling; sac exposed; artery ligatured above and below sac; sac removed; entire; wound closed	Wound healed by primary intention. Recovery complete	The sac was rea dissected up, as t were no adhesi ? Traumatic.

No.	Reference.	Surgeon.	Name.	Sex.	Age.	Locality.	Artery affected.	Other treatment adopted.
2	Hoppe, Über Behandlung der Aneurysmen, Greifswald, 1885	Löbker	—	F.	8	Left popliteal space	Left popliteal artery	Not mentioned
3	Beiträge zur klinischen Chirurg., 1892, vol. ix	E. Kübler	T. R.	M.	56	Right popliteal space	Right popliteal artery	Forcible flexion of leg upon thigh; digital and instrumental compression
4	Ueber Extirpation von Aneurysmen, Inaug. Dissert., Berlin, 1886	Köhler	—	M.	29	Right popliteal space	Right popliteal artery	Digital and instrumental compression
5	Jahresbericht der Chirurg. Abtheilung, Basel, 1891	Socin	Z.	M.	63	Left popliteal space	Left popliteal artery	Not mentioned
6	Bull. et Mém. de la Soc. de Chirurgie, Nos. 9 and 10, 1895	Heurtaux	—	F.	11	Femoral aneurysm, left	Common and superficial femoral	Not mentioned. No disease; traumatism suggested from early infancy
7	Unpublished	Walsham	A. B.	M.	49	Right side of neck, extending from jaw to near clavicle	Common carotid	None

Symptoms.	Operation.	Result.	Remarks.
Pulsating swelling; pulsation very violent, and loud systolic bruit audible	Longitudinal incision over tumour; sac covered by internal popliteal and perinæal nerves; nerves drawn aside, sac dissected up, artery ligatured above and below; the sac removed entire	Wound healed by granulation after being packed with iodoform gauze. Recovery complete	No history of an i could be obtain account for the o rence of an aneu in so young an vidual.
Large pulsating tumour size of hen's egg; usual signs of aneurysm; compression of artery above failed to arrest pulsation	Incision 6 inches long over tumour; sac exposed; artery ligatured above and below sac; sac then extirpated entire	Wound healed by first intention. Recovery complete	The Hunterian o tion was not tho advisable, since pression failed arrest the pulsati
Large pulsating swelling size of goose's egg; skin over tumour discoloured; some œdema of leg and foot	Longitudinal incision over tumour; femoral artery ligatured above sac; anterior and posterior tibials ligatured below; sac extirpated; 2 inches of the popliteal vein which was adherent to the sac was also removed	Wound healed well. Perfect recovery. No untoward result from interference with vein	The whole popl artery was occupie the aneurysm, w necessitated liga of femoral and til
Large pulsating tumour size of goose's egg; had existed 1¼ years	Incision 6 inches long over tumour; artery ligatured above and below sac; sac removed entire; wound drained	Wound healed well. Patient left hospital in 4 weeks	In spite of advanced and therefore gre risk of atheroma the arteries gener patient made goo covery.
Tumour extended from 2 inches below groin to lower part of Hunter's canal. Had been growing 15 months	Esmarch's tourniquet; incision 8 inches long; artery ligatured above and divided two places; sac dissected up; sac removed entire after ligature applied to popliteal; upper part of sac ruptured during operation; alarming hæmorrhage; abdominal aorta compressed; 5½ inches of femoral vein removed	Good recovery	Sac was adherent; 3 lated arterial bran secured and tied ing dissection.
Large non-pulsating swelling; no sign of aneurysm present	Exploratory incision; tumour cut into; enucleation begun; gush of blood. Discovered to be an aneurysm. Carotid tied below with two ligatures, divided between; sac dissected up, and several arteries and main internal carotid tied distal side and sac removed	Wound healed by first intention, except where drain-tube inserted, and at one spot above where some ligatures came away. Recovery complete	Sac fairly easily sected up ex above, where a adhesions; adhe vagus dissected sac. Tumour be operation thought be a growth in gla probably maligna

No.	Reference.	Surgeon.	Name.	Sex.	Age.	Locality.	Artery affected.	Other treatment adopted.
8	Wiener klinische Rundshau, March 15th, 1896	Maydl	B. K.	M.	45	Popliteal space	Left popliteal	None
9	Ibid.	Maydl	W. W.	M.	44	Popliteal space	Left popliteal	None
10	Ibid.	Kopfstein	J. K.	M.	35	Popliteal space	Right popliteal	None
11	Revue de Chirurgie, ix, 1889	Mollière	—	—	—	Popliteal space	Popliteal	—
12	Ibid.	Peyrot	—	F.	50	Popliteal space	Popliteal	—
13	Arch. für klinische Chirurgie, xliv, 1892	Schmidt	G.	M.	30	Popliteal space	Left popliteal	—
14	Ibid.	Schmidt	,,	,,	,,	Popliteal space	Right popliteal	Distal compression; flexion
15	Léon Comte, Thèse de Lyon, 1885, and Archiv für klin. Chir., 1892	Polloson	—	M.	42	Popliteal space	Popliteal	Digital and instrument compression
16	Verhandlungen der deutschen Gesellschaft für Chirurgie, xxi Congr., Berlin, 1892	Rehn	—	—	—	Popliteal space	Popliteal	Not stated
17	Verhandlungen der Berliner medicin. Gesellschaft, Oct. 16, 1895	Israel	—	F.	62	Back of thigh	Third perforating branch of profunda	None

Symptoms.	Operation.	Result.	Remarks.
Pulsating swelling size of hen's egg; usual signs of aneurysm	Esmarch's bandage; longitudinal incision; nerve separated; artery and vein ligatured above and below; sac removed; wound plugged	Wound healed by granulation	Delirium tremens. covery.
Pulsating swelling; usual signs of aneurysm; œdema of limb	Longitudinal incision; nerves separated; artery and vein ligatured above and below; sac dissected out; wound closed	Healed by first intention	Delirium tremens. covery.
Pulsating swelling; usual signs of aneurysm	Esmarch's bandage; longitudinal incision; nerves separated; artery and vein ligatured above and below; sac removed; wound closed	Healed by first intention	Delirium tremens.
Diffused aneurysm; great size; about to burst	Esmarch's bandage; extirpation	Wound healed in 20 days	Complete recovery
Very large; usual signs of aneurysm; œdema of limb	Artery and vein tied above and below; sac extirpated	Wound healed; complete cure	Artery above sac way on applying ture. Complete very.
Very large; about to burst	Esmarch's bandage; longitudinal incision; vein separated; artery tied above and below; sac removed	Wound closed; deep sutures. Operation lasted 3 hours	Easily separated its connections. (plete recovery.
Pulsating swelling	Esmarch's bandage; longitudinal incision; vein separated; artery tied above and below; sac removed	Wound closed; some slight suppuration; healed in 9 days; complete recovery	Difficult to sep vein; vein woun lateral ligature.
Not stated	Vein and artery tied; sac extirpated; wound plugged with iodoform gauze	Wound became septic; knee suppurated; death	Erysipelas, alcoho emphysema.
Not stated	Sac extirpated; artery and vein tied. Details not given	Gangrene; amputation refused; death	Vein wounded, tie
Pulsating swelling in parts; thought might be sarcoma	Esmarch's bandage; puncture; longitudinal incision; sac opened; sac enucleated; third perforating artery led into sac; wound plugged	Wound healed well	Very difficult to rate from great s nerve.

No.	Reference.	Surgeon.	Name.	Sex.	Age.	Locality.	Artery affected.	Other treatment adopted.
18	Annals of Surgery, July, 1898	Moynihan	H. S.	M.	31	Subclavian region	Third portion of right subclavian	Iodide of potassium; rest
19	Wiener klin. Wochenschrift, Sept., 1891	Schopf	—	M.	46	Subclavian region	Subclavian and part of axillary	None
20	Bulletin of Johns Hopkins Hospital, July and August, 1892	Halsted	—	M.	—	Underneath clavicle	Subclavio-axillary	None

Three Cases in which Sac was exposed, Artery tied on Proximal a...

| 1 | Frenken, Inaug. Dissert., Bonn, 1886 | Trendelen-burg | N. N. | M. | — | Right groin, extending up under Poupart's ligament | Common femoral | ? |
| 2 | Progrès Médi-cale, 1891, 2nd series,13,14, p. 346 | Bazy | M. R. | M. | 44 | Inguinal | Inguino-femoral | None (?) |

Symptoms.	Operation.	Result.	Remarks.
Smooth round tumour size of hen's egg, firm, elastic, and pulsating; bruit	Curved incision over clavicle; flap dissected up; clavicle divided in two places and pulled downwards; anterior scalenus retracted; pleura and veins separated; two ligatures applied to second portion of subclavian; artery divided between; aneurysm dissected up; axillary tied; sac removed	Erysipelas; wound infected, but healed up, and all well till 59th day, when second aneurysm formed and bled, on which the common carotid and innominate were ligatured. Patient died 1 hour after operation	Catgut ligature ¹ Extensive diseas¹ arterial vessels.
Soft, elastic swelling size of fist; no pulsation, no fremitus; no radial pulse, no œdema, no cyanosis; pulsation had never been noticed	Exploratory incision; pectoral divided; found to be an aneurysm; artery on distal side already obliterated; proximal vessel tied high up under clavicle; axillary vein separated with difficulty; sac removed	Wound healed by first intention	Sac thick-walled, with firm blood-c
Large, solid, but elastic swelling, overlapping middle third of clavicle; feeble pulsation; thought to be a growth	Subclavian tied in two places above clavicle, artery divided between; aneurysm, with clavicle and piece of deltoid, dissected up, with 6 centimetres of subclavio-axillary vein; distal (axillary) artery ligatured	Wound healed in ideal way	—

Distal Side, Sac then laid open, Clots turned out, and Sac plugg

Large pulsating tumour; pulsation in sac so forcible that rupture seemed imminent	Longitudinal incision over tumour, commencing 2 cm. above Poupart's ligament; sac exposed; external iliac artery ligatured above sac and femoral below; sac then laid open and clots turned out; sac not removed but plugged	Wound healed by granulation; recovery complete	The sac could n¹ readily dissected so it was left *in* ? Traumatic.
Inflamed, œdematous	Incision over external iliac; peritoneum turned back; artery and vein tied above and below; attempt to dissect out sac; hæmorrhage; sac opened and plugged; wound partly closed, partly plugged with iodoform gauze	Wound healed in a month	No after trouble.

No.	Reference.	Surgeon.	Name.	Sex.	Age.	Locality.	Artery affected.	Other treatment adopted.
3	Berl. klin. Wochenschrift, 1891, p. 908	Karewski	H.	M.	30	Neck	Carotid	None

Three Cases in which a False Sac was laid open, Clots turned out, Tr

No.	Reference.	Surgeon.	Name.	Sex.	Age.	Locality.	Artery affected.	Other treatment adopted.
1	Lancet, 1894, vol. ii, p. 1143	H. Little-wood	—	M.	40	Left thigh, 4 inches below Poupart's ligament	Superficial femoral	Medicinal
2	Lancet, 1894, vol. ii, p. 1143	H. Little-wood	—	M.	63	Popliteal space (right)	Right popliteal	The aneurysm having become diffuse no other treatment was indicated
3	Schmidt, Inaug. Diss., Greifswald, 1890	Helferich	—	—	—	Calf of leg	Posterior tibial	Femoral artery ligatured; elastic compression

Symptoms.	Operation.	Result.	Remarks.
Slight fluctuation in places; no pulsation; thought to be a blood-cyst or enlarged glands	Sac exposed; common carotid, external and internal carotid tied; sac laid open; clots turned out; plugged	Healed completely; plug removed on 4th day	Huge size. Only covered to be an ¦ rysm at operatio:

Sac discovered, Artery tied above and below, and True Sac remo

Pulsating swelling size of lemon; pain and tenderness over tumour; systolic bruit, but no thrill; pulsation in both popliteals synchronous and equal. Three weeks later swelling suddenly became diffuse; upper two thirds of thigh discoloured from blood extravasation; lower limb œdematous; no pulsation in arteries below	Longitudinal incision over swelling, and transverse incision inwards for 6 inches; sac laid open, clots turned out; original rent in sac then seen; artery above and below sac ligatured and sac removed; wound closed and drained	Complete recovery. Wound suppurated, and small portion of skin sloughed; healed by granulation	In this case the pa was suffering a thoracic aneur When the fer aneurysm became fuse the entire was threatened gangrene, the p tion having ceas the popliteal and ¦ arteries. The other alternative to amputate thr¦ the thigh high which would prol have been fatal.
Rounded and somewhat diffuse pulsating swelling about the size of half an orange; skin discoloured from blood extravasation; pressure on femoral arrested pulsation	Incision over the whole course of popliteal; sac opened, clots turned out; original rent in sac found on a level with middle of condyles, and opposite to a pyramidal piece of bone attached to the external condyle; artery ligatured above and below sac, and sac extirpated; wound closed with sutures and drainage-tube inserted	Complete recovery. Wound healed without interruption.	This was a case of fuse aneurysm, th being ruptured pressure against spicule of bone. the patient had a 20 years previousl which he injured right knee, the , always remai somewhat stiff a wards, it is prol that the aneurysm indirectly depen upon that injury.
Pulsating swelling; had increased in size since ligature of femoral artery; after elastic compression suddenly became larger and pulsation ceased	Longitudinal incision over tumour; sac exposed and incised; when clots were turned out the sac was found to be adventitious, the original sac seen behind it; the artery having been secured above and below the true sac, this was removed entire	Wound healed by granulation; recovery complete	It is probable that ture of the sac been caused by elastic compre¦ made over the The swelling incre so rapidly in size gangrene of the was feared.

Three Cases in which Sac was opened, Clots turned out, Arter

No.	Reference.	Surgeon.	Name.	Sex.	Age.	Locality.	Artery affected.	Other treatment adopted.
1	Deutsche med. Wochenschr., 1886, S. 82	Sonnen-burg	—	M.	49	Right popliteal space	Right popliteal	Digital compression
2	Deutsche Zeitschrift für Chirurgie, Band xxii, 1885	Scriba	D. A.	M.	38	Popliteal space	Popliteal	None
3	Brit. Med. Journ., vol. i, 1897, p. 1161	Littlewood	—	M.	37	Popliteal space	Right popliteal	None

Four Cases in which Sac was opened, Clots turned out, Arte

No.	Reference.	Surgeon.	Name.	Sex.	Age.	Locality.	Artery affected.	Other treatment adopted.
1	Lancet, 1876, p. 597	Thomas Annandale	A. M.	M.	37	Left groin, just below Poupart's ligament	Common femoral	Compression ligature of external iliac after which pulsation returned
2	Lancet, 1896, vol. ii, p. 599	Mac-gillivray	—	M.	64	Inguinal region	External iliac and common femoral	Ligature of external iliac subsequently, on recurrence of pulsation, ligature of four vessels on distal side
3	Lancet, 1896, vol. i, p. 228	Raymond Johnson	—	F.	12	Scarpa's triangle	Common femoral	None
4	Med.-Chir. Trans., vol. lxiv, 1881	Morris	—	F.	45	Neck	Common carotid	Common carotid tied

ligatured on Proximal and Distal Side of Sac, and Sac removed.

Symptoms.	Operation.	Result.	Remarks.
Large pulsating tumour size of apple; pulsation so violent that rupture of sac feared	Longitudinal incision over tumour; sac exposed and opened, clots turned out; artery ligatured above and below sac; sac removed piecemeal	Localised gangrene of foot; slough separated and wound healed	The gangrene asca to thrombosis of p teal vein found at ration.
Fluctuant in places; no pulsation; thought to be a sarcoma or ruptured aneurysm	Esmarch's bandage; clots turned out; vessels (11) above and below sac tied; aneurysm was arteriovenous; sac extirpated; wound sutured with Czerny's sutures	Wound healed in 22 days after. Death 2 months after operation from tuberculous lung affection	Ruptured arterio nous aneurysm.
Large diffused aneurysm; great pain	Incision; clots turned out; artery tied above and below; sac excised	Complete recovery. Wound healed rapidly	The aneurysm had noticed for 6 wee]

ligatured on Proximal and Distal Side of Sac; Sac not removed

Pulsating swelling having deep attachments, size of hen's egg; all characteristic signs of aneurysm present	Longitudinal incision over tumour; sac laid open, clots turned out; four arteries then seen to arise from interior of sac; each of these was ligatured outside of sac; sac not removed	Wound healed by granulation. Eleven days after operation smart attack of secondary hæmorrhage, 9 oz. of blood lost. Complete recovery	In this case not had compression fi to cure the aneur but ligature of external iliac wai tended with no b result. Patient viously had aneui of right femoral c by compression.
Large recurrent aneurysm	Sac opened; vessels supplying aneurysm tied; sac plugged	Granulated. Perfect recovery	Sac could not be sected up.
Aneurysm, probably embolic; well-marked signs	Sac opened; artery ligatured above and below; sac not removed (?)	Good recovery	External iliac trolled by finger pa through linea sen naris.
Recurrent pulsation and increase in size	Incision; clots turned out; arteries tied above	Recovery	Artery on proximal already obliterate

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. xi, p. 72.)

THE MORBID ANATOMY AND PATHOLOGY

OF

DR. BRADSHAW'S CASE

OF

MYELOPATHIC ALBUMOSURIA

BY

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On the 26th of April, 1898, one of us (Dr. Bradshaw) read before this Society an account of a case in which the urine contained a very large quantity of a form of proteid which was shown by its chemical reactions to be related to the class of bodies known as albumoses (1). The main facts of the case which were then reported, and its subsequent history until the death of the patient, were briefly as follows.

A man, 70 years of age, a respectable shopkeeper in

Liverpool, noticed about the end of the year 1896 that he passed milky urine from time to time. In August, 1897, systematic examinations of the urine were instituted, and it was found that it always contained a large but variable amount of a proteid body which was coagulated by a temperature below 60° centigrade and yielded other reactions which served to identify it with the peculiar albuminous body which was first described by Bence Jones in 1847, and is now commonly known as albumose (2).

Beyond the fact of the presence of this remarkable body in the urine there was at first nothing to suggest that the patient was the subject of any serious disease. All the ordinary indications of renal disorder were wanting, and for some months there was nothing in his circumstances or condition which afforded any clue to the significance of the remarkable condition of the urine. An examination of the literature, however, showed that the kind of albumosuria described by Bence Jones had been observed in six cases in all, and that in every instance it had been found to be associated with an affection of the bones of the trunk. This affection had been at first regarded as a form of osteomalacia, but the more recent observers had found that it was a form of multiple myeloma, a disorder in which the bones undergo absorption, and become attenuated in consequence of the development of a new growth which originates in the marrow spaces and spreads outwards, causing absorption of the hard parts of the bone. In the few instances in which albumosuria had been found in connection with multiple myeloma, the existence of the latter had only been recognised on the post-mortem table, it had never been suspected during the patient's life; but the records of these cases led Dr. Bradshaw to suspect that this might be a case of multiple myeloma, and to watch carefully for the advent of local indications of bone disease. This suspicion was strengthened when, near the close of 1897, the patient complained of severe pains and tenderness

about the ribs, sternum, and back, and it became almost a certainty when, early in 1898, there appeared obvious signs of diminished rigidity of the bones, consisting of an unnatural yielding of the chest walls to the pressure of the hand or the stethoscope, curvature of the spine, and subsequently spontaneous fractures of the ribs. During the progress of the case there were remarkable remissions in the intensity of the symptoms, both local and general. In January, 1898, the patient was unable to leave his bed on account of the pains, and suffered from a severe attack of bronchitis, followed in February by pneumonia affecting one lung and accompanied by rusty sputum. For some days he seemed to be *in extremis*. After this he gradually improved; the pains subsided almost entirely, the lungs cleared up, and in the spring he went to reside with friends in Cheshire, and for two or three months was able to get up and down the stairs and to take short walks out of doors. About the middle of July the pains returned with great intensity, and he was unable to leave his bed ; deafness was noticed on one side, uncontrollable vomiting came on, and he died apparently from exhaustion on August 10th. The peculiar character of the urine persisted till the end, but a specimen passed a few days before the fatal issue contained a little albumin in addition to the large quantity of albumose.

Owing to the death having taken place in a private house some valuable time was lost before arrangements could be made for removing the remains to a mortuary and holding an autopsy. A limited examination only was permitted, but it was amply sufficient to confirm the diagnosis made during life.

The following are the chief points of the necropsy, which was made by us fifty-nine hours after death. There were also present Dr. James Barr, Mr. W. T. Thomas, and several other practitioners who were interested in the case.

Externally there were no indications of decomposition. Rigor mortis was present. On removing the integu-

ment from the thorax a small quantity of subcutaneous fat was found.

The whole bony framework of the thorax, the sternum, ribs, and vertebræ, presented a striking departure from the normal. The bones were so brittle that they could be broken by a slight pressure with the finger, a condition more noticeable in some spots than in others, and there were several complete fractures which had doubtless occurred during life. On the right side the sixth rib was found broken across about three inches from its cartilage, and there was an incomplete fracture of the seventh rib in a corresponding situation. The eighth rib presented a slight irregularity which seemed to be due to a fracture which had united. On the left side the fifth and sixth ribs were broken about half an inch from their cartilages.

The change was very pronounced in the sternum, which broke while it was being removed. The gladiolus contained a large irregular cavity filled with a red mass resembling splenic pulp, which was easily squeezed out by slight pressure on the bone. This cavity corresponded to the position of a spot of great tenderness, which had been especially noticed several months before death.

There was a remarkable backward curve of the dorsal spine, and projection forward of the lumbar vertebræ and the promontory of the sacrum, constituting an extreme exaggeration of the normal curves. The degree of the curvature in the dorsal region was roughly measured by drawing a chord from the front of the body of the seventh cervical vertebra to that of the fourth lumbar. The body of the ninth dorsal vertebra, where the backward projection was greatest, was two inches behind this line.

The body of the seventh dorsal vertebra was removed; it was soft enough to be easily cut with the knife. The cut surface felt soft, and presented a disintegrated appearance not unlike that of splenic pulp.

The organs presented no striking abnormality. Those in the thorax were in their usual position; the pleuræ were free except for some slight adhesions at the apex; the right contained about one ounce of blood-stained serum, the left about two fluid drachms. There was no fluid in the pericardium. The lungs were normal, the heart weighed 8½ ounces; the right auricle contained a partly decolourised clot; the tricuspid orifice admitted three fingers. The left auricle was normal; the mitral orifice admitted two fingers; the valve appeared to be competent, but the curtains were thickened; the aortic valve was competent, and to all appearance healthy; there was slight atheroma at the beginning of the aorta.

Post-mortem changes were much advanced in the abdominal organs, especially the pancreas. The liver weighed 37 ounces, its surface was normal; there was no fatty change in its substance and no secondary growths; the hilum was free; the gall-bladder was filled with bile, and there were no gall-stones. The stomach, intestines, and spleen were to all appearance normal.

The right kidney weighed 2½ ounces. The capsule was rather too adherent, and on removal showed a whitish-yellow cortex mottled by distended capillaries. At the upper end a cyst the size of a hazel-nut was found, containing a glairy greenish fluid. At the lower end there was a similar cyst about the size of a pea. The cortex was much diminished, the pyramids were very white, the blood-vessels were not very obvious. On transverse section the outer third of the organ was seen to be occupied by a denser material of a yellowish-white colour. The left kidney weighed 3½ ounces. On section it resembled the right, but the departures from the normal were less apparent. On the convexity there was a cyst the size of a hazel-nut. The appearance of the organs led us to suppose that they were able to perform their functions fairly well until the death of the subject.

No enlarged glands were found anywhere, nor were

any tumours observed either attached to bones or in any part of the body.

A piece of the upper end of one tibia was sawn off. It appeared to be quite normal.

An examination of the head was not allowed.

The material reserved for further examination consisted of a piece of a rib, of a dorsal vertebra, and of the sternum, some of the gelatinous marrow-like substance in the sternum, and pieces of kidney.

On the same day some of the fresh marrow-like substance was extracted with a weak sodium chloride solution. The extract gave a marked albumose reaction with nitric acid and a turbidity with hydrochloric. Unfortunately the rest of the marrow-like material was by inadvertence placed in weak formalin solution, with the result that the proteids were rendered insoluble, and so their coagulation points could not be taken. As far as it went, the evidence pointed to the soft material in the bones containing the same substance as was found in the urine.

The piece of rib exhibited, preserved in formalin, shows that the bone is reduced to a mere shell about the thickness of note-paper, but its general outline is preserved. The interior is occupied by soft material, which in the fresh state was a bluish-red semi-solid pulp which had taken the place of all except the most superficial parts of the bone (Pl. V, fig. 1).

A small piece of rib, freed as far as possible from periosteum and other soft material, and dried in an incubator, was sent to Mr. Collingwood Williams, the Liverpool city analyst, who kindly undertook the analysis of it for us.

His report is as follows :

The quantity available for analysis was 0·117 gramme.

Organic matter	...	45·85 per cent.
Mineral ...	...	54·15 ,,
		100·00 ,,

Plate V.   Med. Chir. Trans., Vol. LXXXII.
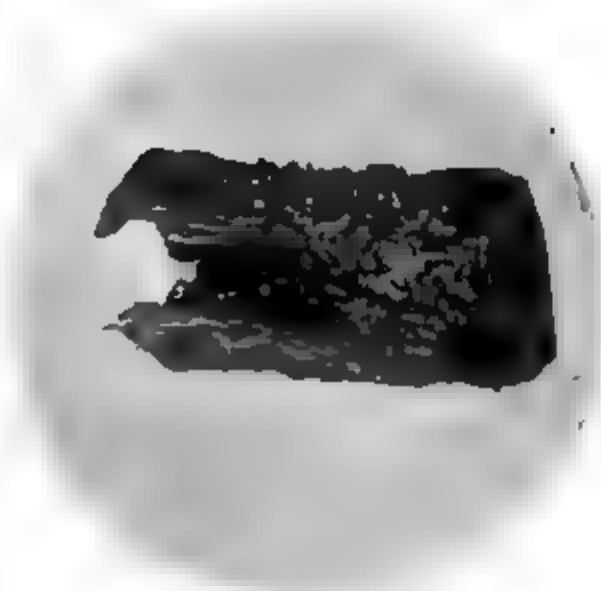
FIG. 1.   FIG. 2.

FIG. 1.—A portion of an affected rib near its cartilage.   To the right is seen the
    cartilage in transverse section; to the left is seen the thin shell of bone, part
    of which has been removed so as to show the contained mass of new growth,
    which has shrunken somewhat and appears dark.   *(Nearly natural size.)*

FIG. 2.—Transverse section of decalcified rib.   The osseous tissue is greatly reduced
    in thickness, and is being invaded by the new growth which is seen *in situ.*
            (Zeiss obj. A without ocular)
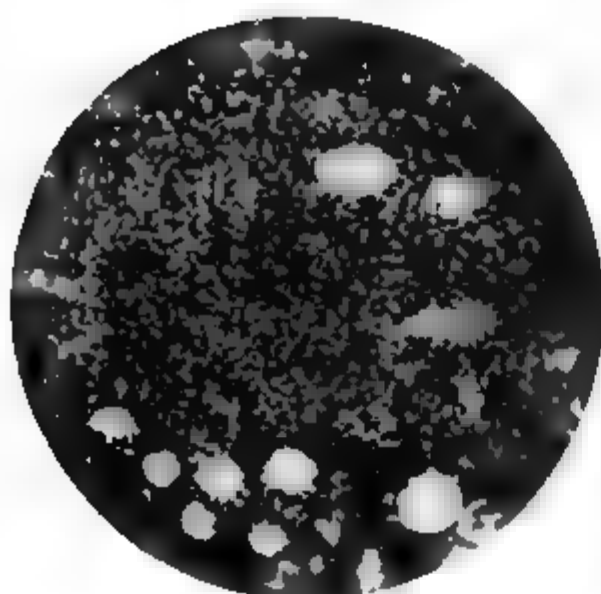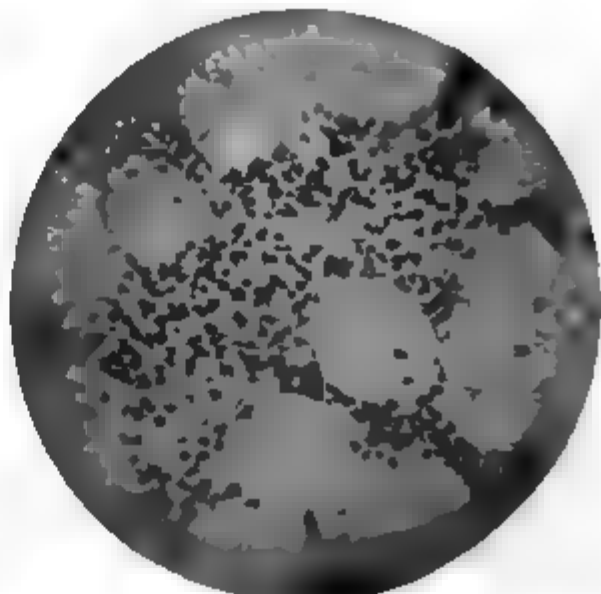
FIG. 3.   FIG. 4.

FIG. 3.—Section of new growth in interior of sternum.   The general characters of the
    structure are those of a round-celled sarcoma. *(⅙ inch objective.)*

FIG. 4.—The same as Fig. 3, more highly magnified, showing the cells of the growth
    in more detail.
    (Apochromatic 3 mm. immersion objective, aperture 1·40, compensating ocular 12.)

Composition of ash :

$$\left.\begin{array}{lr} \text{Ca} \ldots & 36\text{·}63 \\ \text{PO}_4 \ldots & 55\text{·}59 \\ \text{Insoluble in HCl} \ldots & 5\text{·}10 \\ \text{Not estimated} \ldots & 2\text{·}68 \\ \hline & 100\text{·}00 \end{array}\right\}$$

Proportion of CaO, to P_2(

5 : 4

If we make due allowance for the fact that no speci; precautions were taken to remove the fat, the proportic of organic to inorganic constituents does not depa: greatly from the normal.

Microscopical Examination.

The length of time which had elapsed since the deat of the patient made the histological examination in son respects less satisfactory than we should have wished.

A piece of rib was preserved in 4 per cent. form solution, and portion$_\backslash$ of the gladiolus and the vertebra i Müller's fluid.

Thin slices of the dried bones were kindly prepare for us by Mr. J. A. Woods, L.D.S., and were mounted i Canada balsam.

The gelatinous marrow-like substance.—This was studie by means of cover-slip preparations fixed with absolu; alcohol and ether, and stained in various ways, vi methylene blue, eosin, and logwood and eosin; and als in sections,—part of the material was fixed by saturate solution of perchloride of mercury, part in Müller's flui; hardened in alcohol, and embedded and cut in paraffi; The sections were then stained by the above-named dye and also by picro-carmine and borax-carmine.

The marrow-like substance was also studied *in situ* i sections of the bone which had been decalcified by meai of a solution of phloroglucin in 5 per cent. nitric acid.

By these means it was found that the cancellous spac: of the ribs, sternum, and vertebra were densely packe

by a round-celled growth. This growth invaded the bones along the vascular channels, which were considerably dilated, so that islets of bone appeared embedded in the mass of cells.

The cells were almost entirely of the lymphoid variety, a single large nucleus being alone visible in the majority.

A few oval or spindle cells were seen infrequently, but no myeloplaques or eosinophile cells were found. Red blood-corpuscles appeared in considerable numbers, but were never observed to be nucleated. In addition to the lymphocytal form of cell, larger cells with a considerable amount of protoplasm and either a single, double, or reniform nucleus were met with (Pls. V and VI, figs. 2, 7 and 8).

The cover-glass preparations showed the same general features, including the absence of myeloplaques and eosinophile cells, but owing to the presence of fatty and albuminous material they were less satisfactory than the sections.

The Bone.—The decalcified bone of the sternum and vertebra showed normal features, there was no transformation of bone into a lower type of connective tissue, such as has been observed in undoubted cases of osteomalacia, and there was an entire absence of osteoclasts. Dry preparations of these bones likewise showed a normal structure. On the other hand, in the rib, the delicate bony shell, which was cut in transverse section, appeared to have undergone retrograde changes; the normal lacunæ and canals were feebly represented, and where present were irregular in shape and distribution. On the inner surface they seemed to have entirely disappeared, and that part of the bone had a fibrous, and at the same time dotted structure, strongly suggestive of a reversion to a fibrous connective tissue. Indeed, the recognition of the tissue as bone would not have been easy had the facts of the case not been already known (Pl. V, fig. 5).

Owing to the thinness of the bony shell it was impossible to prepare a dry transverse section of the rib so as to compare the inner and outer parts. A dry tangential

Plate VI.   Med. Chir. Trans., Vol. LXXXII.
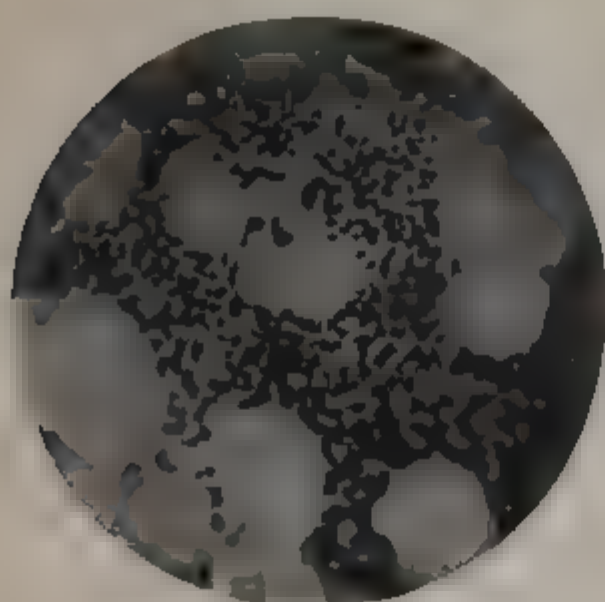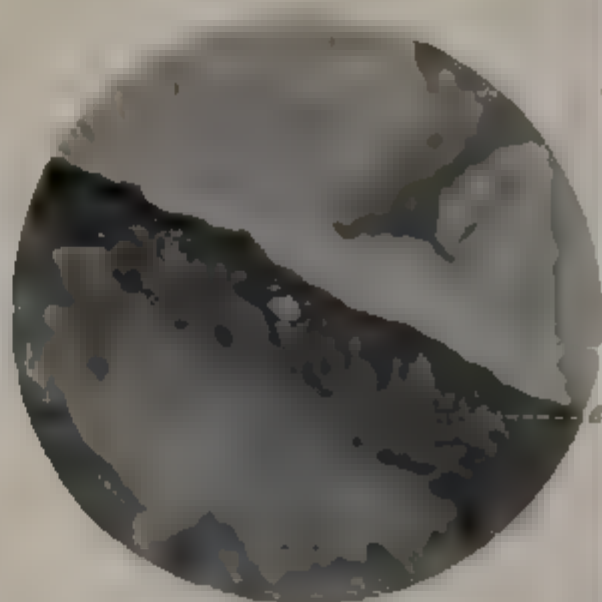
FIG. 5.   FIG. 6.

FIG. 5.—The same as Fig. 4, another part of Section.

FIG. 6.   Transverse section of decalcified bony shell of rib.
    (*i*) Inner aspect of shell, in contact with new growth which has become
      detached during manipulation.
    (*e*) External surface, with muscle attached.   This section gave some
      evidence of a retrograde change in the structure of the bone.
      (⅓ *inch objective.*)
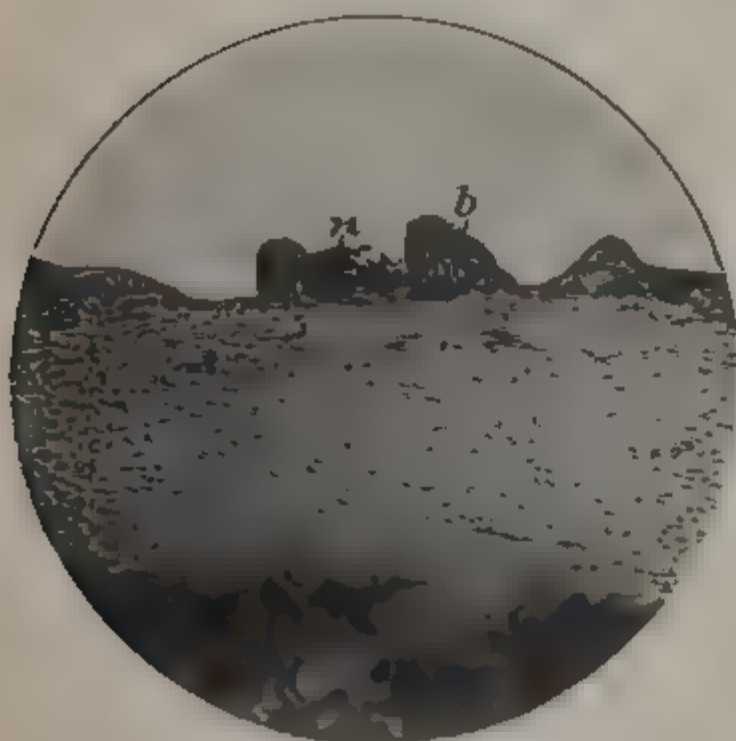
FIG. 8.

FIG. 7.

FIG. 7.—Transverse section of superficial layer of decalcified sternum.
    (*b*) Internal surface of osseous layer, apparently undergoing erosion.
    (*n*) Fragment of new growth *in situ*. (*Zeiss obj. A without ocular.*)

FIG. 8.   Tangential section of dried bony shell of rib, prepared by grinding.   Shows
  normal disposition of lacunæ and canaliculi. (*Zeiss obj. A without ocular.*)

section showed a perfectly normal arrangement of lacunæ and canaliculi (Pl. VI, fig. 6).

Kidneys.—The microscopic appearance was practically that met with in the milder forms of chronic interstitial nephritis; the connective tissue showed at places proliferation, and the cells of the tubules were found to be in a granular degenerated state, and occasionally lying free in the lumen.

Summary of the morbid anatomy.—As far as the limited examination which was permitted enabled us to ascertain, the characteristic lesions were confined to the bones of the trunk, but it is possible that the cranial bones were affected in a similar way. These lesions consisted in a disappearance of the osseous tissue, beginning centrally, and the growth in its place of a soft vascular mass of undifferentiated round cells. The remaining osseous tissue of the rib appears to have undergone retrograde changes, but that of the other bones shows normal characteristics, from which we infer that the primary lesion was the invasion by the cellular growth. There is no evidence to suggest the existence at any time of primary disease of the kidneys, the changes found in these organs being such as can be accounted for by the irritation arising from the passage through them of the abnormal material which they excreted for many months.

It is obvious that the histological characters of the growth in the interior of the bones merely show that it consists of embryonic cells which have undergone little or no differentiation, and are separated by a very small amount of intercellular substance. It might, therefore, be regarded anatomically either as a round-celled sarcoma, a lymphoma, or a granuloma. Clinically, its progressive development and fatal issue seem to entitle it to rank with the malignant neoplasms, but it differs from them in remaining strictly limited to one kind of structure, and not spreading either to contiguous parts or to distant organs. The cellular mass has originated apparently in the cancellous tissue or marrow spaces of the ribs, sternum,

and vertebræ, and by its proliferation has caused absorption of the earthy salts and the proper tissue of the bone.

When we come to consider the pathological relationship of this condition we find that, apart from rickets, which only affects the growing bone, cases of extreme flexibility and brittleness of bone fall into two great divisions. On the one hand we have those in which the essential pathological change is a simple absorption of the earthy salts, the Halisteresis Ossium of some authors, a process which, when it affects the skeleton as a whole, constitutes the disease known as osteomalacia or mollities ossium. On the other hand, we have the cases in which absorption of the hard parts of the bone is due to its invasion by some kind of new growth, a condition which, when it occurs locally, is familiar to surgeons as myeloid sarcoma.

Within the last quarter of a century pathologists have come to recognise a more or less generalised absorption of the bones, arising from the simultaneous development of new growths in the bones in various parts of the body. This condition was first described as a distinct disease in 1873 by von Rustizky of Kiew (3), under the name of multiple myeloma, a designation under which several cases of multiple bone tumours have been recorded since. There can be no doubt that cases of multiple myeloma have been confounded with osteomalacia. The differential diagnosis between the two diseases is probably not always possible during life, but as a rule their clinical pictures are sufficiently distinct. Osteomalacia is almost confined to young women, often coming on in connection with pregnancy. All parts of the body are implicated, the extremities in a marked degree, with the result that the patient is soon confined to bed. The bones are often bent into most fantastic shapes, but actual fractures are few. Multiple myeloma, on the contrary, mostly occurs in men in the latter half of life, the bones of the thorax are those chiefly affected, the patient is able to leave his bed until near the end, deformities are not extreme, and fractures are common.

We have compared the microscopic sections in our cas with sections from a case of undoubted mollities ossiur which occurred in the Liverpool Royal Infirmary in 188 (4). The sections were made by Mr. F. T. Paul, who ha kindly placed them at our disposal. In our case, as w have already shown, the interior of the bone is occupie by a cellular mass which is invading the hard parts. I Mr. Paul's specimens the cavities of the bone are occupie by what seems to be marrow, judging from the presenc of large myeloplaques, and around this we find a layer o dense fibrous tissue, which seems to represent the bon tissue from which the salts have been removed. Briefl stated, it appears that in osteomalacia the bones ar softened owing to loss of earthy salts; in multiple mye loma the osseous tissue is atrophied without any obviou change in its chemical composition. In the one case th bones bend, in the other they break.

The morbid anatomy of multiple tumours in the bone differs in different cases, and the records leave us uncer tain whether the descriptions of multiple myeloma ma not refer to two or more varieties of disease. There is however, one circumstance which serves to differentiat these cases of bone disease into two distinct categories namely, that some were attended by albumosuria an some were not.

In this communication we will confine ourselves t cases in which albumosuria was observed, and of thes only six well-attested instances were on record when th present case was first communicated to this Society i April last year. The leading features of these cases wi now be considered.

I. Macintyre's and Bence Jones's case (5). A descri tion of the urine was communicated to the Royal Societ by Bence Jones in 1847, and the whole case was brougl before the Royal Medical and Chirurgical Society by D Macintyre in 1850.

A man 45 years of age suffered from excruciatin pains in the chest, back, and loins for upwards of a yea

The progress of the disease was not uniform, but striking temporary improvement was noticed from time to time. There was tenderness on percussing the chest, but no deformity was observed. The urine contained the albuminous body which we now call albumose, and gave reactions which had never been recognised before, but which were described in detail in the paper read before this Society last April, and need not now be repeated. There was no suspicion during life that the bones were the seat of any morbid affection.

At the post-mortem examination all the ribs were found to be soft and brittle. They could be readily cut with a knife, and could be broken by a very moderate amount of force. Their interior was charged with a gelatiniform substance of a blood-red colour and unctuous feel. The sternum and the bodies of the vertebræ were similarly affected, but the bones of the extremities were not affected, and the kidneys were healthy.

The microscopical examination of two lumbar vertebræ and a rib was made by Mr. Dalrymple, whose description corresponds very closely with our case (6). He says, " The disease appears to have commenced in the cancellated structure of the bone, for the external osseous laminæ are firmer and more healthy than the internal. The external are still hard, requiring the exertion of some force to cut them ; they are thin, however, and when sliced expose large cancellous cavities filled with a red gelatiniform substance, threaded here and there by fine bony fibres." The bulk of the gelatiniform tissue was composed of nucleated cells, but there were also present granular matter, oil globules, fat cells, caudate cells, and blood-cells. It is worth noting that there is no mention of multinuclear myeloid cells. The condition was regarded as a form of mollities ossium ; but Mr. Dalrymple makes the shrewd remark that it bears some resemblance to a malignant disease of the bone, and in the light of subsequent research we shall hardly be wrong in concluding that this was in reality a case of multiple myeloma.

II. The second case, Kühne's (7), observed in 1867, wa; that of a man aged 40, whose urine contained the bod described by Bence Jones. Deformities and other indica tions of an affection of the bones of the trunk wer observed during life, and the diagnosis of osteomalacii was made; but no post-mortem examination was obtained

III. The next case, which was investigated by Kahle (8) and Huppert, was that of a medical man who died ii 1887 at the age of 54, having been ill about eight years The symptoms were, for the most part, pain and tender ness, and, later, deformities in the bones of the trunk Albumose was recognised in the urine for six years befor the fatal termination.

The post-mortem examination showed the existence o softening of the ribs, sternum, and vertebræ. Micro scopically the condition was found to be :—extreme osteo porosis with formation of numerous Howship's lacunæ determined by the multiple proliferation of a tissue con sisting of rather large round cells (round-celled sarcoma so-called myeloma).

In this case several bony tumours were found in connec tion with various affected bones.

Kahler's paper contains an exact clinical picture o the case. The diagnosis made during life was osteo malacia, but the author suggests that the occurrence o albumosuria might in future serve to distinguish multipl myeloma from osteomalacia.

IV. The next case was under the care of Stokvis o Amsterdam, and was published in 1891 (9). There wa; albumosuria but no indication of bone affection durin{ life. After death, however, the bones were found highl brittle, and the place of the marrow was taken by a re(gelatinous mass.

There were also in the connective tissue, the muscles, th periosteum, and the serous membranes numerous dry firn tumours of yellowish-white colour. Unfortunately n(microscopical examination was made, and it is quit possible that the tumours scattered about the body ha(

nothing to do with the growth in the bones. In this case it is stated that the humerus was affected. The kidneys contained some chalky deposits, but appeared to be normal in other respects.

V. The next case was observed in the clinic of Stintzing of Jena in 1895 (10). The patient was a man aged 61, who presented marked deformity, the back being in a position of extreme kyphosis, and the head sunk between the shoulders. Pains in the back and the chest were severe. The urine was investigated with great care by Professor Matthes (11) of Jena, who came to the conclusion that the albumose which was present was really a body distinct from digestive albumoses, and indeed from all other bodies known to chemistry. The diagnosis made during life was osteomalacia. The *post-mortem* showed that there was extreme softening of the bones confined to the trunk, the enlarged marrow spaces being occupied by a dark bluish-red soft tissue. Microscopic examination of a piece of affected rib gave the following result :

The compact tissue was wanting in places. Where it was present it was very much thinned, and contained numerous wide, irregular lacunæ, filled with a vascular spindle-celled tissue. Beneath the compact tissue, and replacing it where it was wanting, was a zone of tissue consisting of closely packed round cells, intersected by irregularly running bands of spindle-cells. It was of unequal thickness, and projected unevenly into the marrow. In it were some rather large islets of cartilage and numerous necrotic patches, which stained feebly and uniformly, and were free from cells and lacunæ. The microscopic diagnosis was therefore given as chondro-sarcoma.

VI. All the foregoing cases were men. The sixth case was a woman aged 36, who was under the care of Prof. Senator, and died in October, 1897 (12). She suffered from severe pains in the chest and back, and paralysis of one of the hypoglossal nerves. No deformi-

ties appear to have been noticed, and the diagnosis was nephritis with albumosuria. The post-mortem showed the presence of a new formation in several of the ribs, proceeding from the medulla and regarded as a myelogenous round-celled sarcoma. The kidneys were amyloid.

A case reported by Noël Paton and Byrom Bramwell was at one time believed by Prof. Huppert to belong to this class. In the former paper the author (Dr. Bradshaw) expressed doubts as to the correctness of this view, and quite lately Prof. Huppert has abandoned it (13).

In all these six cases the condition of the bones was either altogether overlooked during life, or was considered to be osteomalacia. In Kühne's the absence of an autopsy leaves us in some doubt as to the actual condition of the bones, but it was probably the same as in the others.

It thus appears that our case is the first published case of albumosuria in which the diagnosis of multiple bone tumour was made during life ; and further, that it is the first case in which the diagnosis made during life has been confirmed by the post-mortem examination.

Quite recently one of us (T. R. B.) has been informed by Dr. Reginald H. Fitz of Boston of another case not yet published, in which the diagnosis was made during life and confirmed by the autopsy. A brief reference to this case will be found in a paper read before the Association of American Physicians last May (14).

The records of the six cases we have given in detail show a general resemblance in their morbid anatomy, and this resemblance is shared by the case we have described ourselves. The appearances are by no means identical, and we find in the records of cases where no albumosuria was observed, descriptions of microscopical appearances indistinguishable from those described in the records of the cases we have quoted (15). This is perhaps to be expected, since round-celled formations may undergo various degrees of evolution, as we

observe taking place in various kinds of sarcoma. It seems to us that the mere microscopical characters of the growths do not by themselves form any sound basis of classification, but we maintain that in spite of their observed differences in structure the occurrence of albumosuria entitles these cases to be placed in a category by themselves.

That a morbid process taking place in a region remote from the kidneys should be associated with the presence in the urine for a long period of large quantities of a substance which it does not contain in any other circumstances, is a rare event in pathology. The presence, however, in such a condition of a substance not only foreign to normal urine, but unknown to the pathological chemist in any other connection, is probably without a parallel. The most obvious analogy, though it is only a remote one, is the occurrence of sugar in the urine in association with disease of the pancreas, but in that case we have only to deal with a familiar chemical substance which is known to take part in the normal metabolism of the body. The occurrence of the albumose cannot be looked upon as an accident; when it is found at all it is present for months or even years, and persists until the close of the case. We venture, therefore, to maintain that cases of bone disease with albumosuria must be placed in a different category from those in which this symptom does not occur; that albumosuria with bone disease is a distinct pathological entity, which is probably as far removed from other forms of multiple myeloma as the latter are from osteomalacia. If this be granted, it is desirable to give a name to the condition which will serve to mark its identity; and we offer the term *Myelopathic Albumosuria* as one which expresses the leading features of the disorder without implying the acceptance of any debatable theories as to its nature.

We may be permitted to recall the fact that we owe the earliest recognition of this disease to a Fellow of the Royal Medical and Chirurgical Society, and that it was

before this Society that the completed record of the first case was laid. Half a century elapsed before its occurrence was observed again in any part of the British dominions, and it seemed appropriate that when the second case appeared it should also be communicated to this Society. But though history repeats itself, it never repeats itself exactly, and while the case presented fifty years ago was the first observed, the case which has been presented now is the first in which a correct diagnosis has been made during life and confirmed by examination after death. We venture to express the hope that the association of the Society with the clinical history and the pathology of this remarkable disorder will not end here, but that before the next half-century has passed away one of the Fellows may be in the position to point out the causes which give rise to myelopathic albumosuria, and perhaps to indicate some means of arresting its course.

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(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. xi, p. 81.)

TWO CASES

MULTIPLE CALCULI WERE REMOVED FRO
LARGE NARROW-NECKED SACCULI

MALE URINARY BLADDER

BY

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As a contribution to the surgery of a condition which
is only sparingly illustrated, the two following cases, with
some remarks on the subject, may be acceptable to the
Society. The point it is intended to give prominence to
relates to the treatment of extra-vesical saccules contain-
ing calculi.

CASE 1.—J. R—, a male aged fifty-six years, came
under observation in August, 1892, with symptoms point-

ing to vesical stone, which had extended over two years. The bladder was irritable, and the urine normal in quantity but ammoniacal, purulent, and offensive. The external urethral meatus was somewhat congenitally contracted. There was no history of previous kidney disease, hæmaturia, or renal colic. The neck of the bladder appeared to be elevated, and gave the impression that the prostate was much enlarged.

The patient was placed under an anæsthetic by Mr. Braine, when, on passing a sound, a stone was indistinctly felt. The urethral meatus, being narrow for a lithotrite, was divided, and then the instrument passed easily. A small stone was found and crushed, and the fragments evacuated, but as this was insufficient to account for the symptoms, it seemed not unlikely that more might be concealed or encysted in the neighbourhood of the prostate. The bladder was therefore opened by a median perinæal incision as for lithotomy. The patient, though tall, was spare, and having regard to the character of the urine, it was thought that this mode of access, both for exploration and drainage—if the latter proved necessary—would best meet the case. On the finger being passed through an incision affording ample room, I was at once struck at finding what appeared to be a healthy, normal organ. After a little searching with the finger a small pit or depression situated above the prostate, and somewhat to the left side of the median line was found, and at the bottom of this the rough or rather broken edge of a calculus was distinctly felt. The finger not being long enough to enter the sac, a pair of narrow-bladed curved lithotomy forceps was guided into it, and then several stones were easily withdrawn. After four or five had thus been removed, the sac was thought to be emptied, but on Mr. Braine and Dr. Highet, who kindly assisted me, making pressure upon the abdomen over the region of the bladder, other stones could be distinctly felt being protruded downwards, and were extracted by the forceps in a similar manner. Altogether thirty-

four calculi, varying in size, were removed, and ther was every reason to believe that the sac was emptie(This was tested in several ways. The prostate prove much smaller than was anticipated, its apparent hype1 trophy being in some measure due to the prominenc given to it by the pressure exercised by the calcul When the sac had been emptied of its contents, its exter and immediate relation to the anterior wall of the rectu1 were readily recognised. It must have been capable (holding several ounces of fluid, and by introducing th forceps to the bottom of the cavity, the amount of tissu separating it from the bowel was found extremely smal It would have been very easy to have established a dire(communication between the sac and this portion of th large intestine. There was very little bleeding, thoug the operation occupied twenty-five minutes. A drainag(tube was put into the sac from the incision leading int the bladder, and the subsequent treatment of the cas resolved itself into endeavouring to secure cleanliness an a free outlet for the contents of the bladder, which, (course, included the discharge from the large and probabl suppurating sac connected with it. The patient left th private hospital on the twenty-fourth day, and returne home. The operation wound had not completely healed

Four months afterwards, Dr. Highet wrote me :—" H has improved very much in his general health, and is no' better and more free from pain than he has been for long time. The only thing that troubles him is th urinary fistula, which still continues to discharge. He ha very seldom to get up at night to pass water, and ha gained weight."

In the following year (1893) he was again under m observation, relative especially to the urinary fistula, an various means were adopted to bring about its closur(but though considerable relief was afforded by irrigatio of the bladder, catheterism, and drainage, the woun failed to heal, and continued at intervals to discharge pt and urine.

In 1894 the patient again presented himself for further treatment, his condition, so far as the fistula was concerned, being practically unaltered. With the view of endeavouring to deal with the sac, which was obviously the cause of the fistula, the bladder was opened above the pubes. The sac relative to the bladder was found much the same as on the occasion of the first operation, when the calculi were removed in 1892. Its orifice would just about admit the finger; its extent and relation to the rectum were unchanged. Careful exploration with forceps and syringe failed to discover any further evidence of stone, though in the previous interval phosphatic concretions had escaped through the fistula. A drainage-tube was passed to the bottom of the sac and brought out through the supra-pubic incision in preference to the perinæal, and drainage, aided by the patient's position, was maintained for some weeks. The abdominal wound gradually closed without any obvious change taking place in the perinæal fistula, through which urine mixed with pus continued to escape. It was interesting to observe that though both wounds continued to discharge pus and urine for some weeks, the tendency to heal was far stronger in the supra-pubic than in the perinæal, and was gradually completed in the former in spite of the efforts to maintain drainage through it. The patient died towards the end of 1896, the opening in the perinæum failing to heal.

I received the following report relative to the composition of one of the calculi (which seemed typical of the remainder) from the Clinical Research Association. Thirty-four calculi in all were removed entire, exclusive of the one that was crushed. The largest stone weighed 150 grains, and the united weight of the thirty-four amounted to 11½ drachms. The analysis is as follows:

" The calculus is composed of tricalcic phosphate, calcic phosphate, and triple phosphate (magnesium ammonium phosphate), the first constituent being present in greatest amount. The greater part of the stone was somewhat

soft and friable, the central part being harder and laminated. The latter only differs from the rest in containing a still larger proportion of the neutral tricalci phosphate. No trace of any urate, oxalate, or other con stituent is present. There is no discoverable nucleu other than the somewhat harder core mentioned above."

CASE 2.—A man, aged 50, was seen in July, 1897. He had during the previous year passed by the urethra small, round, and facetted calculi, and as there was some enlargement of the prostate it was suggested, in the absence of any other explanation, that these concretion were possibly connected with this part of the urinar apparatus. The composition of these calculi will be presently referred to. There was no history of any rena colic or any other urinary symptom or disease, and the stones had generally been spontaneously voided withou much difficulty. The prostate was examined from the rectum, and found to be larger and harder than natural especially on the right side. Having regard to the age of the patient and the nature of the enlargement, i seemed almost impossible to form a precise diagnosis o the case, and a wish was expressed to Dr. Hopkinson who asked me to see the patient, for another opportunit of doing so. In the meantime the patient went to Cromer.

About one month after my seeing him he passed a large number of small calculi, which were reported to be composed of calcium phosphate, with a small proportion of calcium carbonate. On this supervened an attack of epididymitis and urethritis. There were no symptom of renal colic or hæmaturia. As he was not emptying his bladder it became necessary to use the catheter, upon which he soon became almost entirely dependent.

After leaving Cromer he continued to pass calcul occasionally, and the urine gradually became offensiv and ammoniacal. He was sounded for stone on mor than one occasion, but nothing was found till towards th

end of 1897, when several small soft calculi were removed by the lithotrite and wash-bottle. About this time evidence was obtained that the bladder was largely sacculated, as it was found possible to catheterise it and a large sac independently of each other, and considerable difference was sometimes observed in the two fluids thus drawn off. It was also noted that when urine was escaping by a rubber catheter from the sac it was possible to arrest the outflow by pressing one finger firmly over a point corresponding in the bladder with a spot about an inch below the right ureter. On pressure being removed the outflow returned, and continued till the sac was emptied.

Towards the beginning of 1898 the patient's condition improved under catheterism and irrigation, and the discharge of calculi ceased for some weeks, so that he was able to attend to his business, and take bicycle exercise. He however continued almost dependent on the catheter, and the urine was usually more or less offensive.

On Easter day, 1898, he took a long and taxing bicycle ride, which was followed that night by most acute pain in the bladder and a severe attack of cystitis, accompanied with high temperature. He returned to town as soon as possible, and I saw him, in consultation with Dr. A. Bland, on April 28th, when acute suppurative cystitis was present. The conclusion we arrived at was that some calculi in the sac had been disturbed by the bicycle ride, and had set up acute inflammation in this confined area, which had extended to the bladder. We saw him again together on May 2nd, and as the symptoms were unabated we advised his immediate removal to a surgical home for operation.

On May 4th an anæsthetic was administered by Mr. Braine, and I was assisted by Mr. Pardoe ; Dr. Bland was also present. The bladder was opened in the usual way by a vertical supra-pubic incision. On examining the base with the finger a small aperture was found, just admitting the index finger, to the right of and a little below the

orifice of the right ureter. The sac with which this aperture communicated contained more than half a pint of exceedingly foul purulent urine and five stones weighing a little over three drachms, facetted, and in the recent state very friable. They were removed with forceps, a matter of considerable difficulty owing to the depth of the sac from the abdominal wound. The base of the sac was pushed up from the rectum, somewhat facilitating their removal; one of the stones was crushed by the forceps during extraction, so soft was it. The sac was flushed out thoroughly with a solution of mercury perchloride 1 in 5000, and a large drainage-tube, inserted to the bottom, was stitched to the edge of the skin incision. Another shorter tube was placed in the bladder and also stitched in position.

The two lateral lobes of the prostate were greatly enlarged on their bladder aspect, rising into the bladder and completely obstructing the internal meatus. It was not considered advisable to remove them at this period, as the bladder was in such a foul condition that septic absorption by the raw surfaces which would have remained would have probably occurred.

The patient's condition was extremely grave for a fortnight after operation. The wound in the abdominal wall sloughed extensively, no doubt owing to the excessively foetid urine which continued to escape alongside the tubes. Various antiseptic solutions were used for flushing, the most satisfactory proving to be a solution of hydrogen peroxide. The patient required dressing at least every three hours during the first ten days. By May 20th the wound was quite clean. The sloughs had all separated, and the edges were granulating in a healthy manner. The pouch was contracting, and would only hold about five ounces of solution. It still secreted a considerable amount of foulsmelling pus. The rubber drainage-tube was removed on this date from the sac, and the latter was washed out twice daily by a full-sized canula attached to an aspirator bottle. After a little practice it became quite easy to

pass the canula into the opening of the sac, although the opening was but little more in diameter than the calibre of the canula. The abdominal wound was allowed to close until only just sufficiently large to admit the canula.

The patient was up and allowed out in a chair. During June several pieces of soft phosphatic stone formed in the pouch and were washed out through the canula; on one occasion a piece required crushing *in situ* by a smooth-bladed lithotrite introduced through the abdominal wound before it could be evacuated. In the last week of June the patient had a sharp rise of temperature, accompanied by several rigors, followed by severe sweating; this lasted several days, and was at length relieved by the passage of about one ounce of most offensive pus.

The sac was evidently not draining satisfactorily, so on July 12th, under ether, the supra-pubic wound was enlarged and the pouch explored again. The opening into it was about the size of a shilling, with very sharp edges. It felt quite circular. The walls of the sac were soft and smooth. A very large vessel was felt beating on the upper and outer wall. There seemed to be a very little tissue between the posterior portion of the sac and the anterior rectal wall. Several phosphatic fragments were removed. The sac was less than half its original size.

The two greatly hypertrophied prostatic lobes were removed flush with the bladder wall by means of cutting forceps. Hæmorrhage was very free, but was checked by firm pressure of pads soaked in liq. ferri perchlor. fort., applied through a caisson. A good deal of oozing occurred during the day, but it ceased in twenty-four hours. On the following day some urine was passed *per urethram*, the first for many months.

The progress of the case was uneventful from this date until his departure from the home. The sac was washed out as before until the abdominal wound contracted. It was then flushed by filling the bladder *per urethram* and by posture (the prone position), emptying it like a bottle into the bladder.

The patient went to Brighton on August 3rd, the urin
then being acid, with a small deposit of pus from th
sac, and mostly passed through the supra-pubic sinus
though some came by the urethra.   He was in Brightoi
under the care of Dr. E. Gordon Barker until Novembe
1st.   Dr. Barker reports that by careful daily washing o
the bladder, with posture exercises (*i. e.* rolling from bell!
on to back and *vice versâ*, knee-elbow position, &c.), th!
sac was kept sweet, but on September 13th he passed *pe-*
urethram a small square piece of phosphatic stone.

He reported himself in London during the first weel
in November on his way to Leamington, where he pro
posed to spend the winter.   He had then gained man)
pounds in weight, and had been swimming and walking
There was still a fistula above the pubes admitting a fin!
probe.   Urine drawn off by catheter was acid and withou
a trace of pus.   Since November he has been under th!
care of Dr. Olive, of Leamington, who writes (January 20th
1899) : " Mr. R.'s condition is much the same as when h!
arrived here.   The thing which troubles him most is th!
quantity of water which escapes through the supra-pubi(
sinus . . . . . the urine remains turbid but is free fron
albumen . . . . . one small calculus about the size of !
large pin's head has been passed since he came to Lea-
mington."

The Clinical Research Association reports that th!
calculi consisted of tricalcic phosphate, carbonate of lime
and magnesium ammonium phosphate, principally of th!
first.

In bringing these two cases before the Society, I an
conscious that the means adopted for the relief of th!
patients fell short of what was desired.   Apart from th!
unusual position the calculi occupied relative to the bladder,
there was nothing, so far as these were concerned, in theii
nature, number, or size, which should have offered an)
obstacle to their successful removal.   Nor was there an)
difficulty experienced in their withdrawal from the sacs
containing them.   It was in the delay, I may say the im-

possibility of obliterating the saccules containing the stones, which eventually led to the death of the patient in one instance and incomplete recovery in the other. I think, however, I may say the latter promises to do well, though the use of the catheter may, as in other instances of urinary disease, prove to be necessary.

When we consider that these sacs are of considerable size, and probably, judging from others that may be examined in museums, of very irregular shape, though their orifices are small, that by reason of their structural formation, they possess no power, either of voluntary or involuntary contraction, and that they are situated between two viscera, the bladder in front and the bowel behind, which are constantly on the move, we cannot fail to recognise the difficulties connected with any surgical attempt to effect their obliteration.

Writing after he had seen the second patient whilst at Cromer, and in reply to a letter which described what had been done, my friend, Mr. Cadge, says, " You seem to have done the right thing very thoroughly, but how can you expect to complete the cure ? I suppose all you can do is to keep the sac well washed out daily. It is, in my experience, exceptional for stones to find their way into sacculi, but in this case, in which small round multiple calculi came down from the kidney, they easily got into the narrow opening which was near the ureter and at the bottom of the bladder which trapped them."

The experience gained from the first case recorded, led me to think, on reopening the abdominal wound in the second case, that it might be found feasible to attempt to substitute a more efficient mode of sac-drainage. This suggested itself, partly by a case read before this Society,[1] where a large hydatid cyst, situated between the bladder and the rectum, was successfully drained from the perineum, and partly by another case not recorded, which appeared to bear more directly upon this point. True,

[1] 'Med.-Chir. Trans.,' vol. lxxviii.

the hydatid case had no direct communication with the interior of the bladder, yet it showed, from one aspect, the feasibility of the proposal.

The other instance was that of a patient who had a sacculated bladder, where the sac spontaneously opened into the bowel, and for some time discharged urine in this way as well as stones. After discharging for some weeks the recto-vesical fistula completely and spontaneously closed, and the patient's condition, so far as his bladder was concerned, has since considerably improved. It appeared to me that if this process had been anticipated by the insertion of a tube into the bottom of the pouch by a perineal incision, it would have prevented the bowel being used for urine-drainage purposes, whilst the result would probably have been equally satisfactory.

Guided by this experience, it was my intention, when I reopened the bladder in the second case, and had removed the hypertrophied portion of the prostate, to have explored the sac, and ascertained whether it was not possible to drain on these lines without interfering either with the urethra in front or the rectum behind. With a pair of forceps in the sac, or any other similar instrument as a guide, there would have been no great difficulty in accomplishing this. However, on exploring the sac with my finger it was obvious that the proposal was not applicable to this case. There was one very large artery, probably an iliac, passing downwards, which could be felt beating from within the sac, whilst a smaller one took a course downwards and outwards. With such relations as these the part could not have been safely approached either with knife or drainage-tube. Though the proposal was abandoned in this instance, I have ventured to refer to it, as the principle might be applicable to other instances. The contractile nature of the neck of some of these sacs, giving the feel to the finger not unlike an elastic band, in conjunction with direct drainage of the pouch below, might bring about a closure of the orifice, as doubtless occurred in the instance

referred to. The original mode of sac drainage through the bladder was therefore reverted to.

There is another point of much interest which I will only mention. Whence are the calculi contained in these sacs derived? Are they primarily of renal or vesical origin, or are they indigenous to the part in which they are found? It is remarkable in both of these instances that there were no symptoms observed which would appear to indicate that the patient had ever suffered from renal colic, though a considerable number of stones were found in each. I am disposed to think they were formed in these sacs.

In concluding these remarks, I would add that I have not intended to include a reference to the pouches or depressions made up of the whole thickness of the bladder wall, but to those herniated protrusions of the mucous membrane between the interstices of the muscular coat, to which the term sac or saccule is now generally applied. The former condition relative to stone has been well illustrated by Mr. Buckston Browne.[1]

In an article on vesical stone, based on 110 consecutive cases which I have recently published,[2] the two instances here recorded were not included, as not being strictly examples of stone in the bladder.

[1] 'Transactions of the Medical Society of London,' 1891.
[2] 'Lancet,' Nov. 12th, 1898.

(For report of the discussion of this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. xi, p. 87.)

ON THE TREATMENT

OF

PULMONARY TUBERCULOSIS BY ANTI-TUBERCULAR SERUM

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THE application of serotherapy to the treatment of tuberculosis was a natural outcome of its great success in diphtheria and other diseases, but the difficulties to be surmounted are of no ordinary kind, the principal one being that in tuberculosis one attack does not protect against a second one, and confer immunity. On the contrary, in tuberculosis one attack generally predisposes to another; and the more numerous the successive attacks, the greater the probability of the system of the individual becoming completely impregnated with tubercle. The form is not always the same. Some-

times the lungs are implicated, sometimes the joints, sometimes the glands, in other cases the intestines and peritoneum, or in others the membranes of the brain become involved, but they are one and all manifestations of the multiplication of the tubercle bacillus and its products or toxins in the individual.

The results of the inoculation of Koch's tuberculin show that the latter is quite sufficient, apart from the tubercle bacillus, to produce disseminated tubercle, and as Virchow's[1] post-mortem examinations proved, to give rise to acute miliary tuberculosis of various serous membranes.

Several medical men have attempted to procure a tuberculous antitoxin by inoculating animals which are reported to be immune from tubercle with that material, and then using the serum of the immune animal for injection. This has been done in France by Richet and Hericourt, by Bouchard and Daremberg and by Bernheim, whose excellent book[2] gives a good summary of work in serotherapy, in Roumania by Babes, and in the United States by Paul Paquin, Trudeau, and Baldwin, the animals used being horses, goats, and dogs, and the fluid employed for subcutaneous injection being sometimes the blood and sometimes the serum alone.

Paul Paquin gives an account of twenty-two poorly-fed phthisical patients, in a condition of more or less advanced disease, treated by injections of serum from an immunised horse, practised once a day for a period of two months, with marked improvement and increase in weight from 1½ to 22 pounds. All were alive five months after the close of the treatment, and about half the number were discharged from the hospital sufficiently well to do their work.

Bernheim[3] obtained an anti-tubercular serum from a goat by inoculating the animal with a cultivation of tubercle bacilli in veal broth, in doses of one to three cubic

[1] 'Berliner Klinische Wochenschrift,' Jan. 12th, 1894.
[2] 'Immunisation et Sérumthérapie,' 1897.
[3] Op. cit.

centimetres daily for five months. The serum thus obtained he described as absolutely inoffensive, and as producing no irritation or fever when injected into phthisical patients.

He claims that he and his colleagues gave, in many cases, from fifty to sixty injections of 3 c.c. doses with good results, selecting the scapular region as the point for subcutaneous injection.

The most extensive experiments on the subject are those of Professor Maragliano,[1] of Genoa, who obtained anti-tuberculous serum by inoculating dogs, asses, and horses with highly toxic principles extracted from cultures of living tubercle bacilli, and then removing serum from the said animals. Professor Maragliano gives the results of the treatment in 412 cases, and indicates what classes of patients are most likely to benefit by it.

While he deprecates any exaggerated expectations in advanced stages of the disease where there exist profound lesions of tissue, and emphatically declares that anti-tuberculous serotherapy can only reasonably be expected to effect a cure in those cases of pulmonary tuberculosis in which no destructive foci exist, he insists that the treatment is applicable to all forms of the disease. He lays great stress on whether the cases are instances of simple tuberculous or of mixed infection, i. e. whether only tubercle bacilli are present, or whether they are associated with diplococci and streptococci, as this association retards and altogether neutralises the effect of the treatment.

It is rather difficult to reconcile this statement with the preceding one, that it is applicable to all forms of pulmonary tuberculosis, and the exclusion of all cases in which diplococci and streptococci exist, would, as all who have worked at the bacteriology of phthisical sputum can testify, restrict the suitable cases to a very small number. M. Maragliano divides his cases into six classes, which

[1] 'Congrès de Tuberculose à Bordeaux,' 1895.

from their mixed nature are somewhat difficult to identify, and claims under the serum therapy, for the first class, presumably the worst (les bronchites avec cavernes), a cure in 7·76 per cent. and amelioration in 37·63 per cent., a stationary condition in 36·55 per cent., and aggravation in 18·29 per cent. In the sixth class the most favourable (broncho-pneumonies circonscrites apyretiques) cure in 66·66 per cent., and amelioration in 27·27 per cent., and a stationary condition in 6·6 per cent. His method is to inject 1 c.c. to 2 c.c. daily, and never to exceed this dose, except in febrile cases, when he gives 5 c.c.

M. Maragliano claims that his serum is (1) completely inoffensive, (2) reduces fever, (3) modifies local phenomena, (4) diminishes the number of the tubercle bacilli in the system, (5) increases the body weight, (6) exercises a useful effect on 91·75 per cent. of phthisical patients, less or more, according to the gravity of the cases, (7) determines a cure in nearly all cases of circumscribed and apyretic tuberculosis, (8) cures even " cavity cases." (9) It may be used advantageously in all forms of tuberculosis.

When M. Maragliano's first paper appeared, we obtained some of his serum from Genoa, and before trying it on patients, we sent a specimen to Dr. Bulloch, the bacteriologist of the Jenner Institute of Preventive Medicine, in order to test its effects on tuberculised rabbits and guinea-pigs. Dr. Bulloch did so, and reported that it was positively inert, and exercised no controlling influence over the march of tuberculosis in these animals.

Under these circumstances we did not feel justified in trying it on patients.

Professors Matucci and Di Vestra give as the result of a series of experiments on sheep, which they selected as the mammal least susceptible to tubercular poison, that perfect tolerance of dead bacillary substance can be arrived at with injection into the blood of repeated and increasing doses of from three to seven milligrammes for every kilogramme weight of the animal. The tolerance of the sheep for living bacillary material was found to be less.

The physiological action of the serum of tuberculised sheep differed remarkably in guinea-pigs and rabbits, the rabbits being far more sensitive to the effects than the guinea-pigs. The mixture *in vitro* of the active serum with the tuberculous virus, in the proportion of not less than four to one, weakened its activity, causing the death of the guinea-pig to occur possibly later than that of the control animals. The mixture, when made in the body of the guinea-pig by injecting into the peritoneum an abundant quantity of the serum before introducing the tubercular virus, caused a more rapid production of the experimental disease.

The serum in question showed no positive prophylactic or curative efficacy against the experimental tuberculosis of the guinea-pig, but a certain slackening of the disease when mixed with the tuberculous virus *in vitro* enabling the animals to survive longer than the control guinea-pigs. After death they exhibited enormous enlargement of the liver and spleen, due in the latter case to hyperplasia of the follicles and pulp, and in the former to infiltration with grey tubercle in the portal spaces. In the lungs there was some caseation, with very few tubercle bacilli. Messrs. Matucci and Vestra find great analogy between the results of these experiments and of Koch's tuberculin, as the original poison of the latter in passing through the organism of the sheep was not much modified. Daremberg, in Strauss's laboratory, injected under the skin and into the veins of rabbits 5 c.c. of serum from dogs who had been tuberculised, at the same time inoculating the rabbits with human tubercle. He found that the preventive serum inoculations, so far from rendering the rabbits refractory to human tubercle, appeared to hasten the tubercular evolution.

Drs. Trudeau and Baldwin[1] made a series of experiments on sheep, asses, fowls, rabbits, and guinea-pigs

[1] "Experimental Studies on the Preparation and Effects of Antitoxins for Tuberculosis," 'American Journal of Medical Sciences,' Dec., 1898; Jan. 1899.

to obtain an antitubercular serum, but failed entirely to protect any of these animals. At the end of their paper is an admirable account of the literature on the subject.

In September, 1895, Dr. Williams had an interview with Dr. Armand Ruffer, at that time Director of the Jenner Institute of Preventive Medicine, with reference to the production of a tubercular antitoxin, and was offered a supply of serum from a horse which Dr. Ruffer had been inoculating with tuberculin, and now had reason to believe had been rendered immune.

The horse in question had been inoculated several times with tuberculin, commencing with small doses and rising to 500 c.c., which amount he had received under the skin on two occasions. A rise of temperature had occurred in the first twenty-four hours after the first injection, but it had subsided, and the animal seemed no worse, and twenty-one days after the last injection a litre of blood had been drawn from the left jugular vein, the clot removed, and the serum separated and mixed with carbolic-acid solution. Though this large quantity of tuberculin had been injected the horse showed no further symptoms, and when we visited him at Sudbury Farm, with Dr. Bulloch, he had a glossy skin and healthy appearance. He was thin, but this was afterwards explained by the condition of his teeth, which did not approximate sufficiently for masticating purposes, and after they had been filed down the animal masticated properly and gained weight rapidly.

During the time he was used for a supply of serum he never showed any signs or symptoms of tuberculosis.

Under Dr. Ruffer's instructions it was arranged that Dr. Bulloch, then Bacteriologist at the Institute farm at Sudbury, should supply us regularly with serum in sterilised bottles of 10 c.c. each, protected from the external air with an india-rubber cap. In accordance with Drs. Ruffer's and Bulloch's suggestions, the bottles were kept in ice, and, as far as possible, the entire contents of

each bottle used up at the time of injection. If a smal portion remained it was thrown away, but if at least hal was left the bottle was quickly closed with sterilizec cotton-wool and the neck of the bottle heated in flame The syringes for injection were by Allen and Hanburys and of the same pattern as those employed for diphtherie antitoxin.

As a rule the syringe, which held 10 c.c., was filled, and injection made into successive patients until the syringe was emptied, care being taken to wipe and sterilize the nozzle after each injection.

Dr. J. Mitchell Bruce kindly placed a ward of foui beds at the Brompton Hospital at the disposal of Dr, Theodore Williams, into which four phthisical patients were placed for serum treatment.

All the injections, with the exception of a few made by Dr. Williams, were carried out by Dr. Horrocks, then Resident Medical Officer, who also kept the notes of the cases, and the frequent sputum examinations were con-ducted by Dr. Perkins and Mr. Hudson, the Assistant Resident Medical Officers. We decided to try the serum in cases of a more or less acute character, where the disease was altogether acute, or where in chronic tuber-culosis acute disease had supervened, our object being to determine what influence a prolonged use of the serum had on the march of tuberculosis, as evidenced by the constitutional symptoms, the sputum, and the physical signs. We also decided later to test it in cases of incipient pulmonary tuberculosis.

Case 1 (Abstract).—Jasper W—, aged 22. Acute pulmonary tuberculosis supervening on chronic disease. Right upper lobe infiltrated and excavated. Left upper and lower lobes infiltrated. Fourteen injections of anti-tubercular serum, given in doses varying from 1 c.c. to 3 c.c., extending over a period of six weeks, caused increase of fever, urticarial rash over body, and swelling of axillary glands. Increase of weight 6¼ lbs. No

reduction of tubercle bacilli in sputum or improvement in the lung condition, but rather the reverse. After the cessation of serum treatment left pleural effusion from fresh tuberculosis, and death by apnœa.

Post-mortem examination showed the right pleura to be the seat of recent pleurisy, and to contain 28 oz. of clear serum, with lymph layer over lung, and lining diaphragm and thoracic wall. The lung itself contained much fibrosis, a number of pigmented tubercles, and two caseous ones at apex. Below apex a shreddy cavity size of a walnut, and below a smaller one. Scattered round some of the fibroid tubercles were some translucent miliary ones; the rest of the lung was partly fibrotic and partly emphysematous. The left pleura was partly adherent, and the left lung was emphysematous, with scattered miliary and fibroid tubercles. A cavity the size of an orange occupied the upper lobe, and communicated with other smaller cavities, all having tough fibroid walls. The heart showed right side dilated and filled with clots; the mitral valve showed on auricular edge rows of small white beads (recent vegetations) varying in size from millet to hempseed, one or two adhering to chordæ tendineæ; weight 11½ oz. A few scattered small ulcers were in lower jejunum and large intestine.

CASE 2 (Abstract).—Ernest K—, aged 22. Acute pulmonary tuberculosis. History four months. Rapid infiltration of right upper and middle lobes, and excavation of upper lobe; infiltration of left upper lobe; fifteen injections of anti-tubercular serum in doses of 1 c.c. to 5 c.c., administered in six weeks; great constitutional disturbance; urticaria around the puncture and swelling of axillary glands. Loss of weight; amount of sputum increased. Number of septic organisms and amount of lung tissue diminished, but tubercle bacilli as numerous as before. Progress of tuberculisation and excavation unchecked by injections.

CASE 3 (Abstract).—William J. C—, aged 23. Chroni tuberculosis of both lungs, with excavation of right uppe lobe. History four years. Twenty-one injections of anti tubercular serum of from 1 c.c. to 8 c.c. in seven weeks followed by local irritation, urticarial rash and swelling and hardening of the axillary glands. Weight unchanged Sputum showed no diminution in number of tubercl bacilli, but reduction in septic organisms and amount o lung tissue. Lung changes showed further excavation.

Blood.—The blood in these first three cases was exa mined microscopically to determine the effect of th treatment on the corpuscles, especially on the leucocytes but with negative result.

CASE 4 (Abstract).—Edgar P—, aged 12. Pulmonar tuberculosis of somewhat rapid development, withou pyrexia. Father and mother both tubercular. Dura tion of illness, one month. Tuberculisation and excava tion of right upper lobe. Tuberculisation of left uppe lobe.

Twenty-five injections of anti-tubercular serum give in doses of from 1 c.c. to 10 c.c. in three months; smal doses better borne than large ones. Injections gave ris to erythematous and urticarial eruptions, and some loca swelling; also to enlarged axillary glands on both sides One injection of 10 c.c. caused lividity and alarming dys pnœa, which subsided, and subsequent injections wel borne. Gain of weight, 4½ lbs. Sputum increased an showed no diminution of the tubercle bacilli, and lun tissue appeared in it under the treatment. Excavatio extended, as evidenced by physical signs and presence o lung tissue.

CASE 5 (Abstract).—James G—, aged 23. Acut tuberculosis of both lungs, the posterior portion of th lungs being affected. History five months. Fifteen in jections of anti-tubercular serum in doses of from 1 c.c to 3 c.c. in fifteen days, and well borne. Axillary gland

on both sides swollen. Weight unchanged. No effects on temperature or pulse. Expectoration increased in quantity, and showed reduction in number of tubercle bacilli and disappearance of streptococci. Tuberculisation extended in the lungs, as evidenced by the physical signs.

Remarks on the first five cases.—The serum produced sundry effects on these patients, two of which were most striking. (1) The urticarial rashes which appeared in four of them, and seemed to be caused by the carbolic acid contained in the first samples of serum, as subsequent ones which contained little or no carbolic acid gave rise to no rashes. (2) The swelling of the axillary glands, generally of both sides, which occurred in all the patients, and followed all the serum specimens.

The effect on the temperature and pulse was rather irritative than otherwise, the expectoration increased in quantity, but there was no diminution in the number of tubercle bacilli, except in one case, though in three cases the lung tissue diminished and in one it increased. Septic organisms were present originally in four of the five cases. They disappeared from two and diminished in numbers in the other two.

The general condition of the patients deteriorated, though two of them gained a few pounds, in spite of pyrexia and severe cough, and at the close of the treatment the physical signs showed that the serum treatment had not in any way checked the ordinary evolution of the disease, but that tuberculisation and excavation had gone on uncontrolled. Experience, however, had taught us two lessons which we determined to profit by in future experiments. One was that our serum was too strong; that it probably had been drawn from the horse too soon after inoculation; and another lesson was that our doses were increased too rapidly. It would appear that tuberculin has the power of passing through an immune animal but little changed, unless it has remained some time in the animal's system, for certainly the effects of

the serum on some of the patients bore a close resem-
blance to those of Koch's first tuberculin. The first
serum was taken twenty-one days after inoculation, and
now we proposed to employ serum taken seventy-two days
after inoculation, to use small doses, and to persevere
with these until complete tolerance of the serum was
established. We proposed also to try the effect on cases
of earlier and more limited lesions, which offered greater
prospects of success. Dr. Biss kindly placed one of his
old wards at Dr. Williams' disposal, to which four
patients were admitted. Two were cases of limited exca-
vation affecting one lung, and two were cases of limited
tuberculisation also unilateral.

CASE 6.—David E—, aged 23, clerk from Port Talbot,
South Wales; admitted into the Brompton Hospital,
April 10th, 1896. He had lost a brother from phthisis
at 27, and had himself had good health up to Christmas,
1895; then cough commenced, accompanied by slight
expectoration, without wasting, night sweats, or fever,
and this continued up to the present time. He had
hæmoptysis (ʒj) at the end of March, but did not feel ill,
and performed his duties regularly, and was somewhat
surprised when a medical man informed him that his
lungs were affected. On admission his cough was mode-
rate, the expectoration scanty, containing numerous
tubercle bacilli, a few micrococci, but no lung tissue;
the temperature 98° F. to 99·5° F.; pulse normal; bowels
regular; urine sp.gr. 1026 and containing no sugar or albu-
men; weight 8 st. 5 lbs. Some dulness, with click sound,
was heard in the upper front of the right chest, and there
was marked shrinking over the lower third of the left front
chest.

Five days later he had sharp pain in the left lower
chest, and friction sound was audible over a considerable
surface. The temperature rose to 103° F. and remained
above 100° F., morning and evening, for more than a

fortnight. On April 23rd the physical signs were the following :—

Right side.—Crepitation in the first interspace, dulness, coarse crepitation, and croaking, cavernous sound above the scapula.

Left.—Crepitation (chiefly extra-pulmonary) over lower half of chest, but well-marked friction sound between the fourth and sixth ribs in the axillary line. The diagnosis was acute tubercular disease of the right upper lobe, undergoing excavation, and secondary.tuberculosis of the left lower lobe, giving rise to dry pleurisy.

The pleurisy gradually subsided, all friction sound and crepitation ceased on the left side, which began to con-tract, the cavernous sounds being well marked in the right supra-scapular region. The temperature fell gradu-ally and reached the normal by May 3rd, and the patient, though much thinner, appeared to have recovered from the recent attack.

May 18th.—There has been no pyrexia for a fortnight, and the physical signs remain the same. Cough is mode-rate. Expectoration ℥ss, muco-purulent, containing numer-ous tubercle bacilli, but no streptococci or lung tissue. Weight 7 st. 10¾ lbs. One cubic centimetre of the anti-tubercular serum was injected under the skin below the right scapula, and was followed by a rise of temperature to 100° F., but by no other symptoms, local or general.

May 26th.—Patient has had four injections of the same amount, given on alternate days. No rise of tempera-ture, but rather a lowering, has followed the last three. The injections were now administered every day, and after the twelfth injection, given on June 8th, as they were well tolerated, the dose was increased to 2 c.c., and continued till June 15th.

June 15th.—Patient improving, and has gained 6 lbs. Cough moderate. The left chest has shrunk considerably, the relative measurements being as following : Right, at level of third rib 15½ in., at ensiform level 15 in.; Left, at level of third rib 14½ in., at ensiform level 14 in.,

showing the left chest to have a smaller circumference than the right by one inch.

The crepitation is less on the right side, and little is to be noted in the right chest save deficient breathing.

. Sputum about ℥ss, containing fewer tubercle bacilli, a good many streptococci, and a little lung tissue. Axil lary glands on both sides have become tender. Pulse and temperature normal. The dose for injection was raised to 3 c.c., and on June 24th to 4 c.c., and on June 26th to the maximum dose, 5 c.c., which was continued till the treatment ceased on June 28th.

June 29th.—Patient has had thirty-two injections, and he has improved. Has gained nearly one stone since the commencement of treatment, weighing now 8 st. 11¼ lbs Cough slight. Expectoration of small amount, and contains tubercle bacilli in considerable numbers, with diplococci, and no lung tissue. Physical signs the same. The axillary glands have quite subsided.

Patient was sent to Sandgate for a month and gained weight and colour. When examined on his return (July 30th, 1896), a dry cavity was still to be detected in the upper right lobe. The sputum was almost *nil*, but contained one to five tubercle bacilli in the field, a very few streptococci, many diplococci, no lung tissue. The total number of injections was thirty-two, extending over six weeks.

Remarks.—This patient improved under the treatment gaining about one stone, and the disease became quiescent. The fibrosis of the left lung probably gave rise to secondary pleurisy, and tended to promote quiescence while the clearing of the right cavity assisted to this end The serum seemed to be well borne, and both local and general symptoms improved.[1]

CASE 7.—John Q—, aged 25, coachman, admitted into the Brompton Hospital, April 1st, 1896. Had led an irregular life, and at one time drank a good deal of

[1] We regret to have to report that this patient died in the summer of 189£ of laryngeal tuberculosis.

spirits.   Was in the British Army for some years, and then in the United States Army.   Has had syphilis, of which no traces are now visible.   Cough came on last August, accompanied by dyspnœa and mucous expectoration, some wasting, and night sweats.   Dyspnœa ceased, but cough persisted.   Signs of tuberculosis of both upper lobes were detected, and a slight diastolic murmur was heard over the cardiac base.   The sputum was examined (April 14th, 1896) and found to contain numerous tubercle bacilli, but no lung tissue and no micrococci.

The temperature was taken every four hours for three weeks and the various symptoms carefully watched.   The temperature was found to range between 97° F. and 99° F., being as a rule subnormal in the morning.   The cough was moderate with mucous expectoration (ℨss. a day) (April 23rd, 1896).   Tongue clean.   Bowels regular. Slight night sweats.   Considerable dyspnœa.   Temperature in afternoon, 98° F.

Right chest.—Slight dulness and crepitation from clavicle to third rib, and posteriorly from apex to about halfway down scapula.

Left chest.—Dulness and crepitation over the first interspace and supra-scapular region.   Weight, 9st. 11lb.

The diagnosis was tuberculosis of right upper and middle pulmonary lobes, without disintegration.   Tuberculosis of left upper lobe of older date.   Ordered injection of anti-tubercular serum—dose, 1 c.c. in subscapular region.

April 24th.—The first injection made to-day, and well borne, giving rise to no symptoms.   It was ordered to be continued on alternate days.

May 6th.—Has had five injections of 1 c.c. each, which have not been followed by rise of temperature or of pulse, or increase of cough.   He complains of deafness, and the right membrana tympani is swollen and injected ; there is evidence of inflammation in the middle ear.   The right axillary glands are slightly swollen and tender.   Physical signs the same.   The expectoration contains a few tubercle

bacilli (three or four in a field), and a few diplococci, but no lung tissue. The ear inflammation having subsided, the injections were continued daily up to May 27th, when the dose was increased to 2 c.c., and thus continued till June 14th.

June 14th.—Patient improving; gained 2 lbs. Cough slight. Expectoration slight, containing two or three tubercle bacilli in some fields and none in others; a few streptococci and numerous micrococci: crepitation less in right lung. Temperature subnormal. Injection to be increased to 3 c.c. Ten injections of 3 c.c. were given, and after the first the swelling of the axillary gland, and especially of the left, recurred, the glands becoming hard and shotty, but not painful. There was also some swelling above the seat of puncture. All these symptoms having, however, subsided, on June 26th the dose was increased to 4 c.c., the maximum reached in this case, and continued till June 29th, when the treatment was discontinued and the patient sent to Sandgate.

. 29th.—Has had fifty injections in doses varying from 1 c.c. to 4 c.c., extending over a period of sixty-six days. The treatment has given rise to no elevation of temperature or pulse. Cough and expectoration, always slight, are about the same. Sputum contains far fewer tubercle bacilli than on admission, and at no time has lung tissue been found in it. The physical signs show great improvement; scarcely any crepitation is to be heard on either side. Patient has gained 5 lbs. since admission.

July 30th, 1896.—Has just returned, after a month at Sandgate, and is well browned and looks the picture of health. Cough very slight, and expectoration almost absent. Scarcely any physical signs to be detected.

January 29th, 1897.—Returned to work after leaving the hospital, but has lately been out of employment and nearly starving; nevertheless he has kept well. Has no cough or expectoration. No physical signs. Started in charge of live stock for Cape of Good Hope, where he was reported as having arrived well and going up country.

Remarks.—In this case the serum gave satisfactory results, as shown in the gradual cessation of cough and expectoration, and in the gain of weight, and disappearance of what were rather extensive physical signs. As long as there was any sputum, it contained tubercle bacilli, though few in number. A good feature, too, in this case was that when the patient fell into bad circumstances and was nearly starved, there was no sign of relapse, and and now that he is living a healthy outdoor life, tending stock on the South African veldt, he has a fair chance of maintaining his recovered health.

CASE 8.—Otto P—, aged 24, single ; a waiter, born in Austria. Was admitted to the Brompton Hospital, April 10th, 1896.

Three years before he had fistula, and was operated on three times, the wound healing quickly, but there still remains a small sinus which does not discharge.

He came to London last year and contracted sore throat, for which he repaired to Ventnor as waiter. Cough and expectoration came on, with some loss of flesh and hæmoptysis (℥iij), and tubercular disease was soon afterwards detected by his medical attendant. The symptoms have continued up to the present time.

On admission, cough and expectoration were moderate. Weight, 11 st. 4½ lb. Expectoration contained fairly numerous tubercle bacilli and a few micrococci, but no lung tissue. The urine was of sp. gr. 1026, and contained no sugar or albumen.

Left chest.—Slight dulness, bronchophony, and crepitation from clavicle to third rib in the supra-scapular region.

The diagnosis was : limited tuberculosis of the upper left lobe without softening. The temperature was taken every four hours for 14 days, and found to range from 97° F. to 99·2° F. Pulse and respirations were normal. Expectoration about ℥ss daily.

It was decided to commence the anti-tubercular serum injections, and on April 24th 1 c.c. was inserted under

the skin below the right scapula, and, as no symptom;
followed, the injections were repeated every other day.

. May 3rd.—He has had four injections of 1 c.c. N(
rise of temperature or pulse ; cough and expectoratioı
slight, the latter containing no tubercle bacilli or micro
cocci.

11th.—Has had seven injections of 1 c.c. each, fol
lowed by no rise of temperature or pulse. Left axillarȷ
glands swollen. Tubercle bacilli absent from the expec·
toration on two examinations, but have now returned iı
considerable numbers, with a few streptococci. Afte:
this the injections were given every day up to May 26th
when the dose was increased to 2 c.c., and continuec
daily till June 14th.

June 14th.—The patient has had thirty-five injections
and shows considerable improvement. Cough and ex
pectoration very slight, the latter containing no tubercle
bacilli or streptococci. Crepitation is less in the lef
lung. The axillary glands are no longer tender. Weigh
about the same. The dose was now increased to 3 c.c
and gradually to 5 c.c.

29th.—Has had fifty injections altogether in abou
nine weeks, with no rise of temperature or pulse, anc
little symptoms except the swelling of the left axillarȷ
glands. Has gained 6 lbs. under the treatment. Oı
the left side of chest dulness and crepitation havɩ
diminished. He says he feels very well. Ordered tɩ
Sandgate for a month.

July 30th.—Just returned from the seaside, well sun
burnt, after a month in the open air. Weight less
9 st. 7¾ lbs. Scarcely any cough. Expectoration stil
present; it contains a few tubercle bacilli, two or threɩ
in field of microscope, and a few diplococci. Tempera
ture always normal or subnormal. Physical signs th(
same. Patient returned to his work.

Remarks.—The treatment decidedly promoted im
provement and arrest of the disease, as evidenced by thɩ
diminution of the cough and expectoration and the de

crease in the physical signs. Weight and vigour were gained.

The sputum at the end of the course still contained a few tubercle bacilli.

CASE 9.—John S—, æt. 25, gas and hot-water fitter, admitted into the Brompton Hospital, July 13th, 1896. Always healthy till January last, when he was attacked with pains in the left side, accompanied by slight cough and expectoration, which have become gradually worse, and by night sweats and emaciation. Expectoration has lately been streaked with blood. Symptoms have continued up to present time. Weight 8 st. 7¾ lbs. Pulse 80. Respirations 24.

Left chest.—Dulness and crepitation over the whole front; crepitation coarse in first and second intercostal spaces, with some tubular sound. Crepitation over the scapular region.

The sputum contains no lung tissue, a few diplococci, and very numerous tubercle bacilli. The temperature was noted every four hours, and found to range from 97·8° F. to 100° F., though this latter point was only reached late in the afternoon. The diagnosis was tuberculisation of the left lung, with excavation in the upper lobe.

1 c.c. of anti-tubercular serum was injected under the skin on July 17th, and the injection repeated on five succeeding days, on the last three of which the dose was increased to 2 c.c., with the result of lowering the temperature instead of raising it.

July 23rd.—Expectoration is abundant, and contains numerous tubercle bacilli and diplococci. After the eighth injection of 2 c.c. the temperature rose to 102° F., but fell gradually in the next three days to 100·4° F., and it was discovered that the patient had a carious tooth and some alveolar inflammation ; the glands of the right axilla were slightly swollen.

30th.—The injections have been omitted for four days,

but as, with the removal of the carious tooth, the temperature fell to normal, they can now be resumed.

August 11th.—Has had seven more daily injections of 2 c.c. each, the temperature remaining within normal limits; then they were omitted until a new supply of serum could be obtained. The first dose of this was given yesterday, and proved stronger in effect than the old serum, for it was followed by a rise to 100·4° F., and the same rise occurred next day after the second dose. The patient gaining weight, and cough less marked.

The injections were omitted for three days, and then resumed with a smaller dose (1 c.c.) which appeared to agree well.

26th.—He has had eight more injections of 1 c.c., and the temperature has only risen twice to 100° F. Patient gaining weight. The left axillary gland still enlarged. Less crepitation in the left lung. Marked flattening of the lower third left chest. Tubercle bacilli and diplococci still numerous in the sputum.

The injections were continued till September 5th, when, as there had been a rise of temperature for three days, they were omitted for five days, and then repeated for seven more days, without causing any pyrexia. The patient appeared to progress favourably. The sputum still contained numerous tubercle bacilli and diplococci.

At the suggestion of Dr. Bulloch, we decided to try the effect of streptococcus serum on this patient, and on September 18th, 5 c.c., kindly supplied from the Institute, were injected under the skin overlying the posterior chest-wall. This was followed by a slight rise to 100·4° F., but no other symptom.

The injection was repeated twice, with a day's interval between each dose, but the two last caused no rise of temperature, or indeed any fresh symptom, and their only influence appeared to be that they were followed by slight swelling of the axillary glands of that side, which swelling gradually subsided. On September 27th the anti-tubercular serum injections were resumed, and

three more of 2 c.c. each were given and well borne, and this terminated the treatment.

October 12th.—Patient has had no injections for fourteen days. Has steadily improved and gained 15¾ lbs. since admission. Cough moderate, expectoration small in amount, nummular, still contains tubercle bacilli, though fewer than before. No streptococci, diplococci, or lung tissue. Breath short on exertion. The left chest shows marked contraction and flattening over the upper half. The right lung is drawn across the median line to the extent of two inches, the heart's impulse is felt in the fourth space, immediately below the left nipple. The stomach is drawn up. Dulness extends from the clavicle to the fifth rib. Cavernous sounds are to be heard to the third rib. Dulness can be detected over the upper third of the posterior chest, sounds dry and clear below. Left chest measures a quarter of an inch less in circumference than the right at the nipple level. The patient leaves the hospital to undertake light work.

Remarks.—The results of the treatment can now be considered. Forty-nine injections in all were given of the anti-tubercular serum, but owing to their causing on a few occasions slight rise of temperature, the dose was not largely increased, as in some of the other cases, and never exceeded 2 c.c. On the whole they were well borne.

The patient's general condition improved, upwards of one stone of weight was gained, and his bodily vigour appeared to increase. Cough lessened. Expectoration diminished, but the number of tubercle bacilli, though very frequent observations were made, cannot be said to have materially diminished ; sometimes they were a few less, sometimes a few more, and they never disappeared. Nor can it be said that the various cocci were distinctly influenced, either by the anti-tubercular or the streptococcic serum. The only result of the streptococcic serum was swelling of the axillary glands of the same side.

The chief beneficial change was in the left lun;
Here undoubted contraction of the cavity, with displace
ment of the adjoining organs, proceeded steadily, and
would seem that this was decidedly promoted by th
treatment.

Remarks on the last four cases.—The effect of tł
serum, as modified, on the last four patients formed
marked contrast to its results in the first five, and thei
seemed to be no doubt as to its beneficial influence, whe
administered in a milder form, and in less severe cases.

The treatment was carried on for longer periods tha
in the first series, and the patients received from thirt;
two to fifty injections, the dose of serum varying from
to 5 c.c., but never exceeding the latter figure. Wheres
in some of the former cases 10 c.c. was reached.

The injections were well borne, and in no case was thei
any local swelling or irritation, or rash either of urticarii
or erythematous form, but in all the swelling of the axi
lary glands of one or both sides took place, as in the firs
set.

The temperature was unaffected in three of the fou
patients, normal or subnormal temperatures bein
generally recorded. In one (John S—) the injection
sometimes caused a slight rise, but no continuous pyrexii

The pulse and respiration were not materially affectec

All four patients gained weight, the excavation case
most, one 12½ pounds, and one a stone; the consolidatio
cases 5½ pounds and 6 pounds respectively. They a
increased in vigour, and were able to return to worł
except Case 9, where a lighter occupation was substitute
for the old one, that of a gas-fitter.

Cough and expectoration greatly diminished in all, an
in one ceased altogether.

All four showed improved physical signs. In the tw
tuberculisation cases they indicated some limitation of th
diseased area; in those of excavation, in one the cavit
became quiescent, and in the other it contracted com
pletely.

With regard to the sputum contents, lung tissue did not appear at all in the course of the treatment as it had done in one of the first cases.

Tubercle bacilli diminished in the consolidation cases and were absent from some specimens, but they were present in others, and were from time to time detected as long as there was any sputum.

In one patient they showed no diminution, though the general progress was excellent. With regard to the other organisms—in Cases 6 and 9, diplococci, staphylococci, and streptococci were all present at first, but disappeared in the course of the treatment. In Case 5 (one of the first series) considerable numbers of diplo-, strepto-, and staphylococci appeared from time to time, but at the end of the injections only a few diplococci remained. In Case 8 a few diplococci were noted throughout. In Case 7 diplococci were always present, and on one occasion streptococci were found.

The two injections of streptococcus serum did not appear to affect the number of tubercle bacilli, but may have had something to do with the disappearance of the other organisms.

It would seem that to obtain an anti-tubercular serum capable of arresting the march of tuberculosis in human beings, the immune animal must have been inoculated several months before any serum is drawn from it and used, for during a considerable period the tuberculin remains unmodified in the animal's system, and the serum of such an animal produces the same effect as tuberculin itself. On the other hand it would appear that when serum is drawn from the horse some months after its inoculation with tuberculin and injected hypodermically into patients suffering from limited lesions of pulmonary tuberculosis, the result is satisfactory, and great general and local improvement follows, which may even proceed to arrest, as in Case 7. The effect of this milder form of anti-tubercular serum in cases of acute or advanced phthisis was not tried, but it is doubtful

whether it would have been much more successful than the stronger form.

. It would be advisable, however, to continue the trial of the milder form in early cases, or those of limited tuberculisation or excavation. The exact mode of action of the anti-tubercular serum is not known, but its influence on the local lymphatic system, as shown by the swelling of the axillary glands, which occurred in all our cases, is pretty evident, and in the lung it appears to promote fibrosis.

In conclusion, we have to thank the Jenner Institute of Preventive Medicine for their liberality in supplying the serum free of cost, and for the able assistance given us at all times by their officers.

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ON PREVESICAL HERNIA

WITH THE

RELATION OF A CASE IN WHICH SUBACUTE STRANGULATION OCCURRED.

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CASES of prevesical hernia appear to be sufficiently rare to warrant me in the relation of a case lately under my observation, and also in an attempt to draw some general conclusions as to the etiology of the condition from it, and the few cases which have been previously published.

My chief object will be to draw a sharp distinction between cases of true prevesical hernia, and those of so-called pro-peritoneal hernia, or " hernie en bissac." Up to the present time (since the paper by Parise (1), the first observer of an uncomplicated case, and the author of the name of the condition), cases of prevesical hernia have been included under the same heading as the true pro-peritoneal cases, and regarded as of the same essential nature. In making this statement, however, I must mention that Krönlein (2), in his criticism of a case of

Baer's (3), and another quoted by Baer and Linhart of a retro-cæcal hernia existent together with an inguinal. hernia, does give some support to what he styles the diverticular theory, and suggests the names of hernia diverticuli, parieto inguinalis, resp. cruralis, for cases in which a common abdominal opening does not exist for the two sacs.

Before proceeding to the relation of my own case, and some other published ones more or less resembling it, I will quote the theses laid down by Breiter (4) in his paper, which completes the essays previously published by Krönlein, and shall then be in a position to indicate in what particulars true prevesical herniæ differ from the commoner pro-peritoneal variety.

In pro-peritoneal herniæ :

1. The hernial sac has two loculi, one situated in the inguinal or crural canal, and a second within the abdominal cavity.

2. The internal loculus lies between the peritoneum and the fascia transversalis. The layer of peritoneum, forming the wall of the sac directed towards the abdominal cavity, blends with the area of parietal peritoneum against the outer surface of which it is applied, so as to form one single layer.

3. The outer sac may be interstitial.

4. The two sacs communicate by a common aperture with the general abdominal cavity.

5. No constant proportion exists between the capacity of the two sacs ; it suffices to class the hernia even if the outer one be quite small.

The details of my own case are shortly as follows :

H. H—, male, a tailor, aged 41. The patient, a healthy and strongly-built man, had had no previous serious illnesses, but eighteen months before he came under observation he developed a right inguinal hernia, this was of the acquired variety and unaccompanied by any abnormality in the position of the testicle. He wore no truss, although the hernia descended into the scrotum, and, in fact, had

taken little notice of the rupture as it gave rise to no inconvenience.

Five weeks prior to his admission into St. Thomas's Hospital, after getting wet through, he was seized with a violent attack of abdominal pain, accompanied by retching. The pain was most acute in the right iliac fossa, but at the same time he suffered with pains over the whole body.

He was seen by Mr. Randolph of Milverton, who found him suffering with abdominal symptoms, distension, pain, and rapid pulse. The right inguinal hernia was down but not strangulated.

To obtain relief from pain he had a way of violently and roughly pushing up the hernia, and this he did frequently.

When seen again, there were vomiting and symptoms of obstruction, and Mr. Randolph thought of "reduction *en masse*" and took him into the Taunton Hospital. The patient remained in the hospital three weeks, during which time he was treated by rest, low diet, and the application of poultices to the swollen abdomen. The bowels were constipated and had to be moved by enemata; for some time previous to the attack there had been some difficulty in getting them to act.

He left the hospital apparently well, both pain and swelling having disappeared, but three days later a similar severe attack of pain in the right iliac fossa, accompanied as before by general swelling of the abdomen, occurred. On this occasion the hernia was not down, there was neither sickness nor constipation, but a considerable degree of fever. He again improved rapidly, and when the pain and fever had abated the abdominal tumour was found, and he was sent to St. Thomas's Hospital.

On his admission into the medical ward the man had few symptoms beyond slight pain in the right iliac region, attended by tenderness, resistance, and rigidity of the abdominal wall. The bowels acted after the administra-

tion of enemata and castor oil, and after a week he was placed on full diet.

No definite opinion could be formed as to the condition of the right iliac fossa, owing to the persistent rigidity of the abdominal parietes, so my colleague Dr. Hawkins had the patient placed under the influence of an anæsthetic on September 18th. A hard nodular swelling was now discovered, most prominent in the right iliac region, but dipping into the pelvis, and extending across into the left iliac fossa. The tumour was hard, and felt much like an ordinary fibroid of the uterus except for its fixity; rectal examination confirmed the presence of the pelvic swelling, but revealed no evidence of inflammatory induration on the right side of the pelvis. After consultation the patient was transferred into a surgical ward, and on September 26th an abdominal section was performed.

A median incision was selected as most suitable to the position of the swelling, and an opening three inches in length in the linea alba immediately above the pubes revealed the fact that the tumour was practically extraperitoneal, the reflexion of the peritoneum being raised at least half the distance between the pubes and umbilicus. A number of large veins lay on the surface of the swelling, which was tense and elastic, and as the possibility of an old hydatid naturally occurred, a small trocar and canula was passed into it. The result of the puncture being negative, the incision was prolonged upwards, and the peritoneal cavity opened to allow of freer exploration. The upper surface of the tumour was found to be peritoneum-clad, extending into the hollow of the sacrum, and evidence of past peritonitis was revealed by the adherence of two coils of small intestine to its central part. *Beyond the adhesion of the intestine, the right extremity of the great omentum was so firmly attached to the tumour that it seemed like a direct continuity.*

On gently introducing the hand into the cavum Retzii it was found that the tumour readily stripped out, so it

was decided to remove it. In order to gain more space the right rectus was now separated from its pubic attachment, a catheter was passed into the bladder to decide the exact position of that viscus which was found displaced backwards and to the left, and the tumour was rapidly stripped out of its pelvic connections and delivered. It now only remained to separate the adherent small intestine, divide the peritoneum along the back limit of its reflexion on to the tumour, and to ligature and divide the omental stalk to set the tumour free. The enucleation was easily carried out, accompanied by very moderate hæmorrhage, and except for the enigmatical mode of connection with the great omentum, there seemed little difficulty in regarding the tumour as one developing in the subperitoneal tissue of the cavum Retzii.

The divided rectus muscle was now sutured, as also the abdominal wall, in layers, but no attempt was made to shut off the general peritoneal cavity as a considerable defect in the pelvic peritoneum had resulted, and the absence of any oozing fortunately rendered this not a matter of great moment.

The after history of the case was uneventful ; the urine had to be drawn off on the first day, but later no difficulty with either the bladder or the bowels occurred. On the fourteenth day a small stitch abscess formed at the lower angle of the wound, but on no occasion did any rise in the general temperature occur. The patient left the hospital on the twenty-ninth day, and has since remained in excellent health.

The right inguinal hernia persists in the same condition as before the operation, and seems to have been in no way influenced by it, and the descent of the bowel is so readily controlled by a strap from his pelvic belt that the patient is unwilling to have the rupture interfered with.

On removal the tumour in outline somewhat resembled a hydronephrotic kidney, the left extremity being somewhat the larger. It measured seven and three quarters

inches in transverse diameter by five and a half in depth from above downwards.

The surface was mottled in colour, yellow and purplish red, resembling a piece of marble of those tints; about a sixth of the whole surface area was covered by peritoneum, elsewhere it was rough and shreddy, and the general appearance suggested recent inflammatory infiltration.

On section a large irregular cavity containing bloody fluid and some old clot was disclosed; this space was mainly occupied, however, by an irregularly branching mass of fibrous tissue highly loaded with lobulated fat.

In the light of a true conception of the nature of the tumour it may be described as follows :

The sac is of reniform outline, but somewhat asymmetrical, the left enlarged extremity which forms the fundus being the more capacious of the two. It is peritoneum-clad for about one sixth of its whole surface area, over the convex margin opposite the hilus. The remainder of the external surface is rough and shaggy, corresponding with the portion torn out of its areolar connections to the pelvic wall and organs (Fig. 1).

When opened the wall is seen to be thick and the interior is evidently lined with peritoneum where free from omental adhesions. The contained omentum appears to have been originally arranged in radiating folds, but as the result of inflammation these are now adherent together, and towards the fundus several large ball-like masses are seen.

The omentum enters by a rounded neck about three-quarters of an inch in diameter, situated at the posterior aspect of the small or right extremity of the sac, at nearly its highest point. The opening is, however, completely closed by the adhesion of the omentum to its circumference. A small secondary sac is seen on the anterior surface (Figs. 1 and 2).

The condition of the contained omentum, and the nature of the bloody fluid evacuated on section, in fact,

exactly resembled that often seen on opening a large, old, irreducible umbilical epiplocele, the omental stalk, as is often the case, having undergone considerable condensation and atrophy as the result of continuous pressure and tension.

On removal of the tumour my first impression was that it offered an example of a subperitoneal fatty tumour similar in nature to those occasionally met with in the subperitoneal tissue elsewhere, as behind the anterior abdominal wall, or between the layers of the broad ligament. The presence of the irregular cavity containing bloody fluid was, however, opposed to this view, and Mr. Shattock, when he saw the section of the tumour, at once raised the question of a retro-peritoneal hernia; to him, therefore, I am indebted for being put upon the right track in determining the nature of the case.

Regarded from this point of view the case may be rapidly disposed of. The patient had, no doubt, been for years the subject of a prevesical epiplocele, which became subacutely strangulated, giving rise to the symptoms observed at the first attack of pain, and rightly attributed to a hernia, although, as the sequence proved, not to the reducible right inguinal hernia observed. Rest in bed, &c., allowed the urgent symptoms to subside, only to reappear as soon as the man resumed his ordinary mode of life, but this time in a somewhat less acute form.

The first attack was probably responsible for the adhesion of the contained omentum to the sac wall, and its subsequent irreducibility, and I am the more inclined to adopt this theory from the fact that in many recorded cases of pro-peritoneal hernia, the strangulation at the neck of the sac has been noted as slight, or as due rather to a sharp bending than to actual constriction, and the contents have been found adherent to the wall of the sac.

The henceforth irreducible nature of the hernia by fixation of the lower end of the great omentum no doubt rendered it much more liable to undergo tension on movement or distension of the abdominal viscera; hence the

speedy recurrence of signs of strangulation on the re-sumption of ordinary life after the first attack.

One point of great importance is unfortunately incapable of a direct answer from observation at the time of opera-tion. In ignorance of the nature of the condition with which I was dealing, I neglected to determine the relation of the neck of the prevesical to that of the inguinal sac, hence I can only offer the following presumptive evidence in favour of my belief that a common abdominal opening did not exist.

The contents of the external sac were readily reducible without affecting the signs of strangulation, while those of the prevesical sac still exhibited signs of strangula-tion.

The prevesical sac was removed, together with its neck and contents, without producing any obvious change whatever in the inguinal hernia, which has continued to descend in the same way as before.

It seems evident, moreover, that the retching and constipation observed depended on pulling on the omentum and stomach, and on direct pressure upon, and obstruction of the rectum, and not on incarceration of bowel. Beyond this also the general symptoms at the time of the attacks, and the adhesion of the small intestine to the peritoneal surface of the upper aspect of the sac found at the time of operation, show that a degree of plastic peritonitis capable of producing these symptoms had been present.

With this evidence in favour of the distinct opening of the two sacs, I should regard the prevesical sac as due to a hernial protrusion of the peritoneum into the middle inguinal pouch, between the deep epigastric and obliterated hypogastric arteries (in fact, in the position usually taken up by a direct hernia). The protrusion commencing in this position, being successfully resisted by the strength of the conjoined tendon, took a further subperitoneal course transversely, superficial to the obliterated hypo-gastric artery, and entered the cavum Retzii, where it

gradually attained the dimensions met with at the time of operation.

The external hernia I regard as a coincidence, possibly the result of the same conditions as favoured the development of the internal pouch, but absolutely distinct from it at its neck.

I will now give a short *résumé* of five cases more or less resembling my own.

The first case (5), that of Parise, almost exactly coincides; it differs only in the absence of any external hernia, and in the fact that, as far as is known, it gave rise to no inconvenience during life, and was only discovered on the postem-mortem table.

In June, 1845, a municipal guard, killed by a gunshot wound, was brought to Val-de-Grace. The body was that of a strong man of tall stature, about thirty years of age, and somewhat stoutly built.

The abdomen having been opened by a crucial incision, it was found that the great omentum was drawn towards the right side, and engaged in a hernial opening about the level of the internal abdominal ring. This opening, a typical hernial neck, was situated in the middle inguinal pouch, the epigastric artery external to it, and the obliterated hypogastric artery to its inner side. The opening was rigid, not puckered, capable of admitting the middle finger, and occupied by a piece of non-adherent omentum. The direction of the opening was outwards and upwards, its inner margin being adherent to the obliterated hypogastric artery which held it back.

The sac, in place of entering the inguinal canal, passed transversely inwards in front of the bladder, behind the lower attachment of the recti muscles ; its fundus crossed the external border of the left rectus, and lay about the level of the left internal abdominal ring.

The contents consisted of omentum alone ; this was healthy, but matted at the neck of the sac, and gathered up into two masses, arranged one to the right and one to

the left of the bladder. In spite of the swellings thus formed, the omentum could be drawn out of the sac without any incision of the neck.

There was no sign of external hernia either on the right or the left side of the body.

In commenting on this case, Parise remarks : " This epiplocele had probably given rise to no signs of importance, but if a loop of intestine had happened to become strangulated in it, what would have been the signs of such an occurrence ? " The case already related offers a partial answer to this question asked forty-four years ago.

The second case, that of Hernu, I quote also from Parise (6).

This occurred in a man about forty years of age, who had suffered with a right inguinal hernia since early youth. The hernia descended to the centre of the scrotum, and for many years had been neglected and supported by a bandage only. In attempting to lift a heavy weight seven days before he came under observation, the rupture descended to the bottom of the scrotum, but it remained soft, and with the exception of a small part of its contents reducible.

In spite of these characters, signs of strangulation developed and persisted, and on the seventh day an exploratory operation was performed; the sac proved thick and indurated, but only a mass of omentum in good condition was found within it, the latter being easily reducible, as a finger could be passed through the ring at its side. Three parts of the presenting omentum were removed, and the remainder returned without any incision of the ring. The omentum had, however, scarcely been returned when it reappeared, and a pad had to be applied to retain it within the belly. From this moment the patient was relieved, and the persistent signs of obstruction were referred to the pull of the omentum on the colon and stomach. The bowels began to act at once, and during the remainder of the day and

during the night a large quantity of fæces, at first hard, then liquid and bilious, were passed.

The relief afforded, however, was short lived. On the following day the patient recommenced to hiccough and vomit, the belly became again swollen and tender, and the condition was worse than before.

The dressings were removed, and a part of the omentum was found to have again escaped; hence it was thought wise to incise the neck of the sac, and a finger was introduced into the belly, but no further information was gained. Further attempts to get the bowels to act proved fruitless, and the patient died on the fourth day after the operation, or the eleventh after the commencement of symptoms.

At the autopsy general purulent peritonitis was found, and beyond the sac already alluded to a second sac, formed by a fold of the peritoneum, situated behind the internal abdominal ring of the same side, and extending inwards between the pubes and the bladder. The second sac contained a loop of reddish-brown intestine, gangrenous in spots, and adherent to the wall of the sac. The two sacs combined formed a " besace," which, attached at its centre to the horizontal ramus of the pubes, descended on the one hand by the inguinal canal to the bottom of the scrotum, forming the external sac which contained the omentum, and on the other descended between the pubes and the bladder, forming the internal sac, which contained the intestine. The aperture of entry of the latter was almost at the level of the internal abdominal ring; it was hard and rounded like a ligament, and the loop of intestine in order to enter the sac was necessarily sharply reflected over the margin at almost an acute angle.

The remaining three cases are quoted from the paper by Krönlein (7) already referred to.

Baer's (8) case. Male, aged 52. Strangulated right scrotal hernia, probable simultaneous co-existent internal strangulation; herniotomy.

The patient had suffered for some years from pain in the right half of the scrotum, this being from time to time severe, and accompanied by swelling in that region. Four days previous to coming under observation, great swelling of the scrotum, accompanied by severe pain and evident signs of strangulation, developed after a strain.

A thick, hard, cylindrical swelling, dull on percussion, was discovered in the right half of the scrotum, the skin of which was œdematous and rigid from inflammatory infiltration.

A herniotomy discovered no hernial sac in the scrotum, but showed the swelling to consist of a much enlarged spermatic cord. High up in the inguinal canal a soft reducible hernial sac was palpable, but was not opened.

Three days later the patient died with all the signs of peritonitis; this was confirmed by the autopsy. Examination of the right lower abdomen and inguinal region revealed the existence of a double hernial sac, the smaller loculus of which, about the size of a walnut, projected into the internal abdominal ring, forming the sac which had been felt during the course of the herniotomy. The larger one was situated in the pelvis against the urinary bladder, it was of general rounded form, and of sufficient capacity to accommodate two fists. The two sacs were in free communication, and a common opening, about the size of a kreutzer, led from them into the general abdominal cavity. The anterior and posterior sacs embraced the horizontal ramus of the pubes, and thus exactly fulfilled the conditions of a "hernie en bissac." The posterior sac lay between the peritoneum and the pelvic fascia, and contained about a foot and a half of small intestine which showed manifest signs of incarceration. The spermatic cord was greatly swollen and hard, and, where exposed in the wound, dry and black.

Streubel's (9) case. Male, aged 73. Double reducible inguinal hernia, with signs of obstruction, possibly connected with the rupture of the right side. The herniæ

had existed some years and a truss had been worn, but on the right side only.

He had been suffering for seven days with vomiting and pain in the abdomen, and when examined was in a state of complete collapse. The belly was enormously distended, the left hernia evident and easily reducible but on the right side no sign of the rupture was dis-coverable. The inguinal canal was open, an empty her-nial sac being palpable in it, and there was no pain or tenderness in the region. The patient said he had re-duced the right hernia seven days previously. The collapsed state of the patient was held to contra-indicate operation, and on the ninth day after the commencement of symptoms he died.

At the autopsy a reducible inguinal hernia containing free coils of small intestine was found on the left side On the right side a large empty sac, extending by the dilated inguinal canal and internal abdominal ring to a spacious cavity lying upon the pubic bone and extending to the bladder, with the upper and anterior wall of which it was intimately connected. This sac was situated between the peritoneum and fascia transversalis, and measured three inches long by two inches from before backwards. Its abdominal opening was situated behind and above, about an inch above the internal abdominal ring at the margin of the psoas, and was about the size of a five groschen piece. In its interior was a knuckle of small intestine about four inches long, black, and almost devoid of polish. The inner margin of the open-ing seemed particularly responsible for the strangu-lation.

Richter's case (10). Male aged 54. Replaced left inguinal hernia; internal strangulation ; herniotomia in-terna.

The patient had a left inguinal hernia of several years standing, and for several days had suffered with symp-toms of obstruction, although the hernia was readily reducible.

Herniotomy showed the inguinal canal to be free, but the spermatic cord swollen; hence the case was regarded as one of internal strangulation; nothing further was done, and the patient died five days later.

At the autopsy the obstruction proved to be due to the incarceration of a coil of small intestine in a peritoneal diverticulum which extended behind the inguinal canal in the direction of the internal ring towards the vesical region, with an opening into the general abdominal cavity a little behind and below the internal ring, that is to say below the opening of the inguinal hernia. The two combined resembled a *hernie en bissac*. In the combined neck of the two the intestine was firmly strangulated, if not already gangrenous.

In commenting on these six cases, I should separate my own and that of Parise from the remainder, placing them in the same category, *as true prevesical herniæ*; that is, as herniæ into a peritoneal pouch independent of any inguinal sac.

The remaining four cases come under the headings laid down in the theses of Breiter already quoted.

The distinction is probably of more genetic than practical importance, but none the less in regarding the two groups some differences may be noted both in the morbid anatomy and symptoms.

Both the prevesical herniæ were epiploceles, in each the sac was of large proportions and occupied a position directly across the front of the bladder. In neither had its presence given rise to any unpleasant pressure symptoms. Thus in my own case there had never been any disturbance of the power of micturition, in spite of the displacement of the bladder; and constipation from pressure on the rectum had, at any rate, never assumed sufficient importance to lead the patient to obtain medical advice.

The internal sacs of the four pro-peritoneal herniæ were of lesser size, and somewhat more deeply and laterally situated, in fact approaching to the condition in

which the internal sac dips down into the pelvis directly behind the pubes. All were enteroceles.

In all four the strangulation was of an acute character, and this, I think, is possibly to be accounted for by the greater fixity of the common abdominal aperture, due to the anchorage afforded by the external sac.

With regard to mode of development my own case offers little support to the old mechanical theory of backward pressure from a truss, or other obstacle to regular descent such as a retained testis, still advocated by Krönlein for pro-peritoneal herniæ; but Mr. Randolph's account of the patient's treatment of the external hernia on the occasion of the first attack of strangulation may be regarded as of etiological significance. I think, however, that the fact that this forcible manipulation of the hernia was carried out as a mode of relief of pain from which the patient had never suffered before deprive it of any serious import. The case is also opposed to the contention of Baer, that the external sac is a diverticular protrusion of the anterior wall of the pro-peritoneal one. The manifest proximity of the two openings in my case, however, separated probably by the deep epigastric artery alone, brings the condition into a somewhat close resemblance to the case reported and discussed by Baer and Linhart, in which a peritoneal gutter had developed between the openings of an inguinal hernia and a retroperitoneal pouch situated behind the cæcum.

Mr. Macready (11), in discussing the mode of development of such pouches, speaks as follows:—"By what means these internal pouches are formed is not apparent, nor have I met with any explanation of the process sufficiently plausible to be reproduced here."

The theory I advance for the development of my own case, viz. that of an interrupted and diverted pouch starting in the same position as a direct inguinal sac, seems however to me to explain at any rate the course taken by the sac, though it naturally leaves the cause of the original commencement of the hernia in the

obscurity which enshrouds the genesis of such a large proportion of ordinary acquired inguinal ruptures.

In conclusion I would sum up as follows :

I. True prevesical herniæ are similar in nature to the so-called retro-peritoneal variety, forming in distinct peritoneal pouches independent of the development of previous or subsequent inguinal ruptures.

II. They bear no relation to congenital herniæ, or to abnormalities in the descent of the testicle.

III. In these characters they are distinctly separable from the pro-peritoneal herniæ of Krönlein, which latter are commonly congenital, connected with vagaries in the descent of the testicle and tunica vaginalis, the sacs resembling in their general arrangement those met with in the various forms of congenital hydrocele; while if in acquired pro-peritoneal hernia the sac descends into the pelvis, this lies rather to the side of the bladder than in the prevesical space.

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Plate VII.   Med. Chir. Trans., Vol. LXXXII.
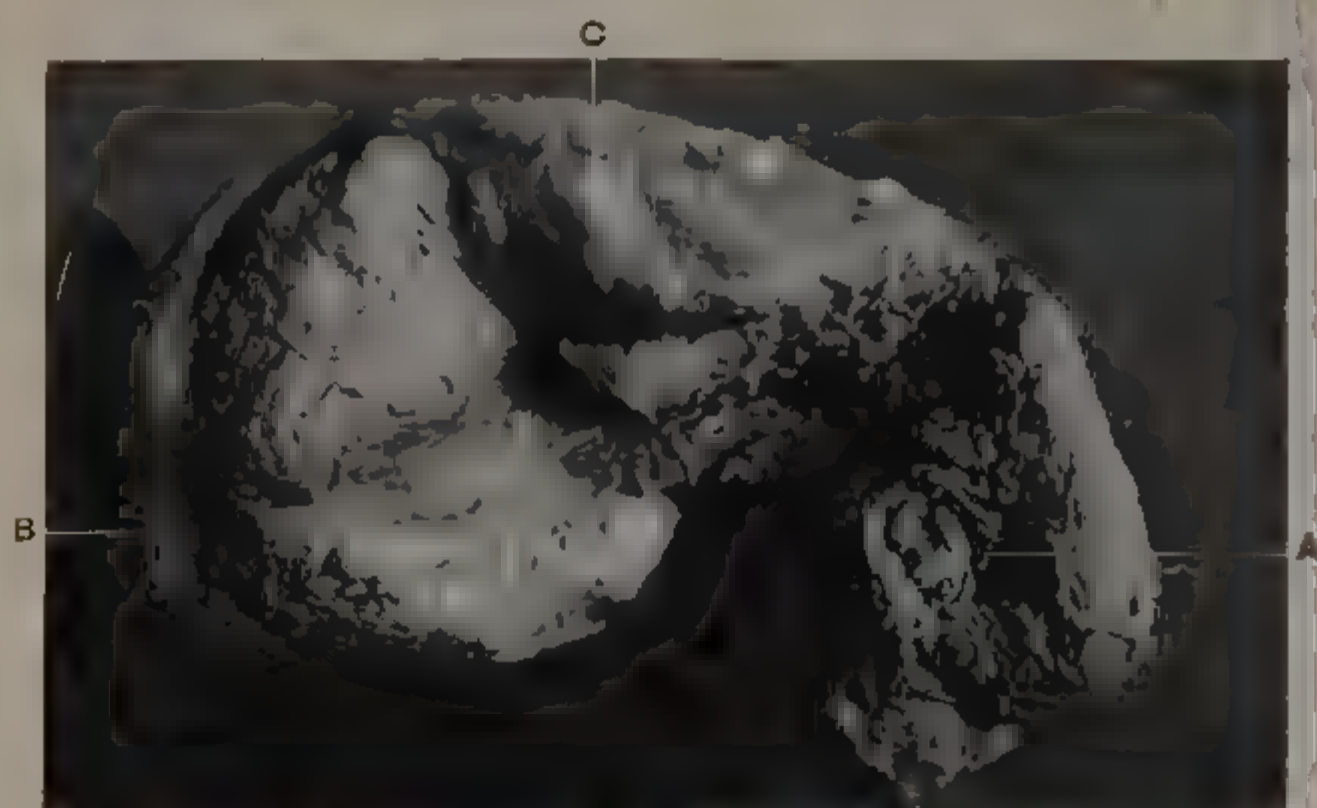
FIG. 1.   Prevesical Sac   External Surface viewed from behind.
A Neck of Sac.   B Fundus.   C Peritoneal clad area.
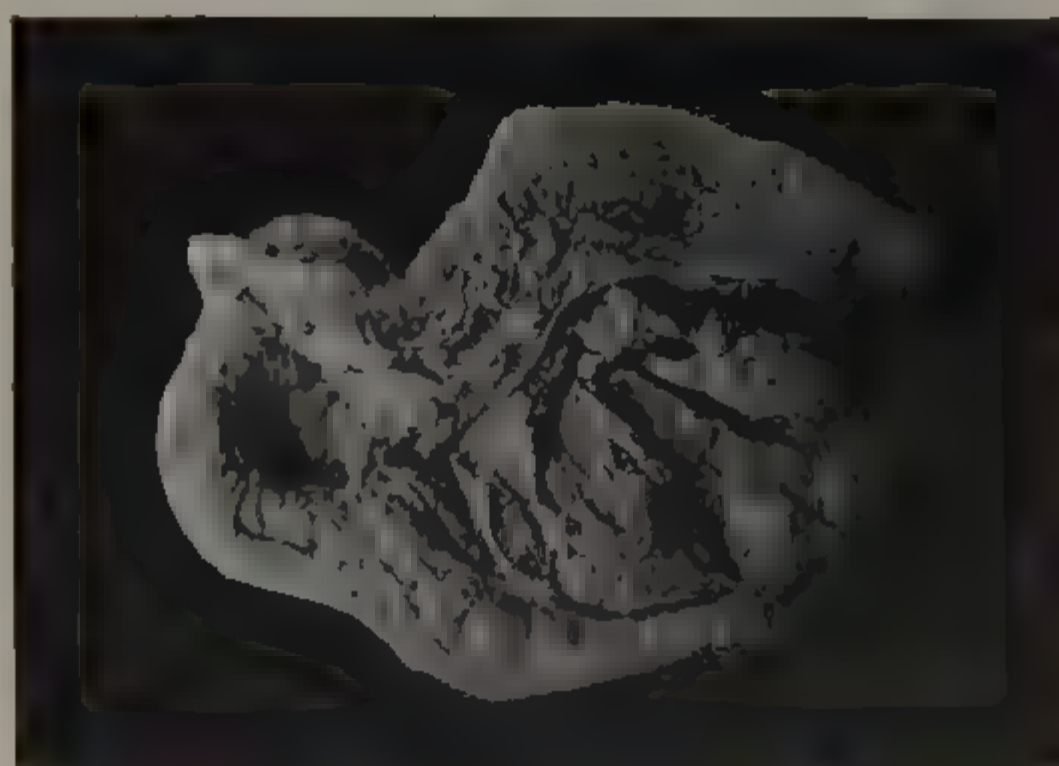
FIG. 2.   Prevesical Sac laid open; the spaces around the
matted and adherent omentum were filled with bloody fluid
at the time of operation.

APPENDIX.

ON PREVESICAL HERNIA, AND ITS TREATMENT BY MEDIAN LAPAROTOMY.

By J. HUTCHINSON, JUN., F.R.C.S.
SURGEON TO THE LONDON HOSPITAL.

THE case now recorded is an example of strangulation of small intestine in a pouch alongside of the male bladder, and might therefore be more strictly termed para- than prevesical, but already too many terms are used with reference to this rare form of internal hernia, which is characterised by the sac lying in immediate contact with the bladder, and by the almost invariable presence or history of an external hernia in the same patient.

This latter point is of importance in explaining the origin of these curious sacs, which can hardly be due to any fact in the development or normal anatomy of the bladder, such, for instance, as is found with regard to the peritoneal fossæ in the neighbourhood of the cæcum and duodenum.

STRANGULATION OF SMALL INTESTINE IN A POUCH ALONGSIDE
OF THE BLADDER; MEDIAN LAPAROTOMY; SUTURE OF
THE NECK OF THE SAC; RECOVERY.

A man, aged 35, came up to the London Hospital on
December 19th, 1898, having suffered for about eighteen
hours from abdominal pain, with, at first, vomiting. For
seven years he had worn a truss on account of a right
inguinal hernia, but he was positive that the hernia had
not come down during the last seven weeks. He was at
work on December 18th, and that evening, without
apparent cause, the lower part of the abdomen became
suddenly painful, and he vomited six times during the
night. Then the sickness ceased, and he had a free
evacuation of the bowels without pain; he came up to
the out-patient department about mid-day, and was
admitted by my colleague Mr. Dean. When I saw him
in the afternoon the region of the right groin above
Poupart's ligament was distinctly tender, and there was
perhaps slight fulness corresponding to the upper part
of the inguinal canal. With the tip of the invaginating
finger a softish swelling, not really in the canal, but
bulging against it, could be felt. There was no general
distension of the abdomen, and no sickness during the
last eight hours, but the suspicion that some internal
obstruction still existed in connection with the former
hernia led me to urge the patient to submit to an
immediate exploratory operation. A true *reductio en
masse* was excluded by the fact that there had been no
descent of the hernia for seven weeks, and therefore no
attempt at taxis had been made during this time by the
patient or by a medical man. Except the tenderness
about the inguinal canal, and the slight bulging felt by
the invaginating finger, together with the man's anxious
expression, there were no symptoms on which to found a
diagnosis.

On the evening of the patient's admission I per-
formed abdominal section in the middle line below the

umbilicus. A ring was found immediately to the right
of the bladder leading down into a small pouch behind
the pubes; a loop of small intestine was tightly nipped
in this pouch, resting directly against the bladder. The
cæcum lay immediately behind the normal position of
the internal inguinal ring, which was empty, and the
vermiform appendix lay parallel to Poupart's ligament,
with its apex close to the neck of the sac.
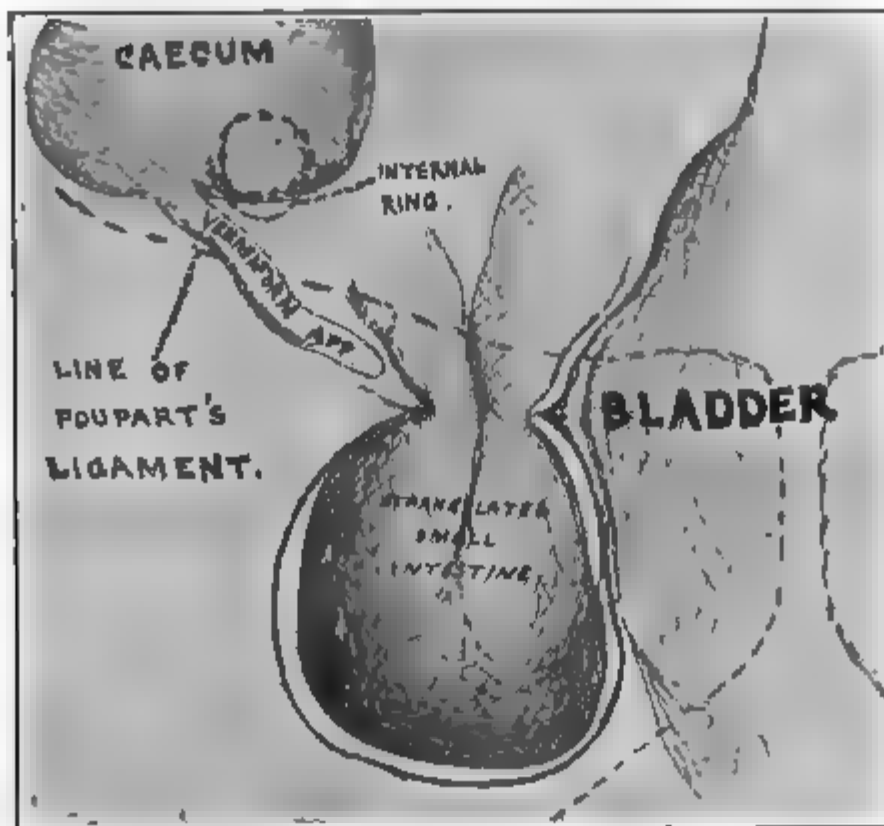
Diagram of prevesical hernia from sketch made at the time of operation.
The neck of the sac is seen to be placed horizontally behind the
pubes and close to the bladder. The relative position of the inguinal
canal and cæcum to the hernia is indicated.

The ring of peritoneum which nipped the intestine
was very accessible through the median wound, but
would have been extremely difficult to reach if the
inguinal canal had been slit up by the latter route. The
intestine was released after notching the margin of the
orifice with a hernia knife, and the sac was found to be
about two inches in depth. With a view of preventing a
recurrence of the strangulation the margins of the orifice
were sewn together with catgut, and the operation then

completed in the usual manner. The wound healed by
first intention, and the patient left the hospital about
three weeks later.

So far as I can ascertain, this is the only case of the
kind operated on at the London Hospital during the last
twenty years.

The following points, suggested by the case just
recorded, deserve consideration :

1. *The relation between the prevesical sac and the
external hernia.*—It seems to be an almost invariable
rule that the formation of the intra-abdominal sac is
preceded by or accompanies an external hernia, usually
inguinal, on one or both sides. In the remarkable case
recorded by Mr. Makins there was a history of a right
inguinal hernia, and of probably vigorous taxis having
been applied. Nevertheless, Mr. Makins contends that
" true prevesical herniæ are formed in distinct peritoneal
pouches independent of the development of the previous
or subsequent inguinal ruptures." The evidence seems
hardly to justify this conclusion, though certainly the
idea that all these cases are due to *reductio en bloc* of
hernial sac and contents cannot for a moment be held ;
equally false would it be to contend that some are not
due to vigorous taxis and displacement of part or the
whole sac within the abdomen.

The occurrence of *reductio en masse,* though probably
becoming rarer every year, is proved by some hundreds
of recorded cases. In one which I had the opportunity
of dissecting some years ago, a hernial sac with its
strangulated contents had been pushed down into the
pelvis between the bladder and the pubes.

One of the earliest recorded examples of this is given
in the ' Clinique de Dupuytren,' vol. iii. A man aged
55 years, had for several years a left inguinal hernia,
which after reduction would stay up for several months.
Strangulation came on, and the symptoms persisted
after vigorous efforts at taxis by a medical man had
apparently reduced the rupture. Six days later M.

Dupuytren performed herniotomy, there being then some intestine present in the inguinal canal, which was certainly not strangulated. The operator discovered what he took to be a tight band within the abdomen, and liberated some more intestine, but the patient died of peritonitis two days later. At the post-mortem it was found that the supposed band was really the neck of a sac placed alongside the bladder, and opening into the general peritoneal cavity close to the internal inguinal ring.

In many cases, as was clearly proved by Mr. Bryant and Mr. Birkett, the reduction has been effected through a rent in the neck of the hernial sac ; and the resulting pouch, although perhaps becoming perfectly smooth, is not really lined with peritoneum. An example of this where the sac was of exceptional size and extended right across the pelvis, pushing the bladder and uterus backwards towards the sacrum, was recorded by the late Mr. Hulke (4).

The prevesical pouch contained no less than eight feet of inflamed small intestine, and a small portion of this had originally become strangulated at the femoral ring. The operation, which had included division of Poupart's ligament, had enabled the surgeon to detect some abnormal condition within the abdomen, but as no median laparotomy was made the true nature of the case was not made out until after the patient's death.

Besides these two forms of prevesical hernia due to vigorous taxis, there is a third variety, in which the sac is really double, and these, as Krönlein has pointed out, are often instances of imperfect descent of the testis.

A great many cases could be cited, the internal pouch being either interstitial or prevesical. One of the first records of this form is given by Leneveu in 1837 (1). A man died with symptoms of internal strangulation, at the time of death he had a freely reducible inguinal hernia on both sides. At the post-mortem, below the orifice of the left inguinal hernia (that is at the internal ring) was found an old hernial sac, reaching down behind the pubes

in which a loop of intestine was strangulated at the neck.

In a fourth variety, of which the case recorded by Parise and the one I have now brought forward are examples, there is no question of *reductio en masse* or of any obstacle to the descent of the external hernia. In Parise's case there was no history of any external hernia, in my case the external hernia had given no trouble, and had not once descended for seven weeks before the onset of the obstruction. It may, however, be urged that even in these rare cases there is a relation between the internal or prevesical hernia, and an external one. It is well-known that the bladder not infrequently lies in close relation with the internal inguinal ring, and that part of it may descend into either an inguinal or a femoral hernia.

I have myself met with this in three or four herniotomy operations, and if in performing radical cure of inguinal hernia by torsion of the sac, the surgeon makes much traction on the latter, he may quite easily draw out part of the vesical wall. We have also positive evidence that sometimes a hernial sac retreats into the abdomen, and of this I could not give a better instance than a case of lumbar hernia described by me in the ' Path. Trans.' for 1889. During life intestine could readily be made to protrude into the hernia, and could be reduced with a characteristic gurgling sound, but after death no peritoneal pouch was present in the mass of fat protruding through the abdominal wall, though it could easily be pushed into it. Arguing from these two facts, the occasional presence of the bladder in a hernia, and the mobility of the peritoneal pouch in a hernia, I would suggest that occasionally the bladder may draw inwards such a pouch, and thus predispose to the development of a hernia within the pelvis.

The following case bears out this suggestion in a striking manner :

M. Havage (' Bull. Soc. Anat.,' 1878, p. 559) records a

fatal case of paravesical hernia, in a man aged fifty-eight, who had worn a truss for some fifteen years on account of a reducible right inguinal hernia. Symptoms of intestinal obstruction slowly developed during eleven days, and the patient died of peritonitis in the hospital, without an operation having been performed. At the post-mortem a fatty hernia was found in the inguinal canal, and within the peritoneal cavity a small hernial sac was present, containing an inflamed loop of small intestine, not very tightly nipped; this intestine had, however, perforated. The neck of the sac was situated between the obliterated hypogastric artery and the bladder, and the sac was situated between the bladder and the pubes, in the subperitoneal tissue. The testis on each side had only descended as low as the external ring, and was atrophied.

In the case just quoted, if we believe M. Havage's statement, there must have been a peritoneal sac in the inguinal canal before the formation of the prevesical one, and the fact that a fatty hernia was found after death, to some extent confirms this (see illustration of lumbar hernia, ' Path. Trans.,' 1889, p. 118, and papers on Fatty Herniæ, Ibid, 1888, p. 451, and 1886, p. 451). A small pouch thus drawn inwards may engage intestine as in the cases recorded by Havage and myself, or omentum as in the cases brought forward by M. Parise and Mr. Makins. There may be no limit to the size that such a hernia may ultimately attain.

2. *What is the best treatment for these cases of pre-vesical hernia?* I think it will come to be generally recognised that median laparotomy gives the patient by far the best chance in these and allied complications of external hernia. In searching the records of pre-vesical hernia one is struck most by the fact that if an operation was done the surgeon contented himself with exploring the inguinal or femoral canal, and that the patient was allowed to die unrelieved when a median abdominal incision would soon have revealed the true

condition of affairs, and in all probability have saved the patient's life (see cases narrated by Hernu, Streubel, Hulke, Richter, Dupuytren, &c.). It is true that sometimes a prevesical or properitoneal hernia may be successfully dealt with without a median incision.

Max Oberst (5) reports a case thus treated, although, since the incision was made only through the inguinal canal, great difficulty was experienced in recognising and relieving the strangulation.

The patient was a labourer aged 35, who had for eight years been troubled with a left inguinal hernia, only partially kept up by a truss. Symptoms of partial obstruction led to repeated efforts at taxis. The patient when admitted to the hospital at Halle presented a swelling in the inguinal canal, and marked fulness above Poupart's ligament. Further attempts at taxis merely increased this bulging of the abdominal wall, vomiting and distension came on, and an operation was undertaken. The inguinal canal was incised, and after easy reduction of some intestine a large sac was detected, the neck of which was situated some 6 cm. above the position of the external ring, and which contained strangulated gut. The narrow opening in this internal sac was gradually dilated with the finger, and the intestine thus liberated. An atrophied testis was situated below the external ring. The scrotal portion of the sac was stitched to the skin, the wound drained, and the patient recovered after an illness complicated by delirium tremens. In reading the account of this case by Oberst, it is obvious that a return of the hernia either into the inguinal or the properitoneal sac had been in no way guarded against by the operation, and that its difficulties would have been diminished if a median section had been carried out. How hard it is to recognise and relieve the internal strangulation through the ordinary herniotomy wound is proved by Krönlein's collection of twenty-three cases, *all fatal*, since the publication of which, however, instances of successful operation (chiefly with the aid of median laparotomy)

have been recorded by Rossander, Neuber, Krönlein, Trendelenburg, and Bolling.

Disappointing on the whole as are the results of abdominal section for acute intestinal obstruction, no cases are more favourable for operation than those of internal strangulation following external hernia. I have myself operated on three such cases, and all recovered. One was found to be strangulation of a Littre's hernia at the internal inguinal ring, the second was a case of strangulation in a wholly detached ring, and the third has just been narrated. With regard to *reductio en masse*, the evidence afforded by Mr. Birkett's classical paper (3) is most striking. He quotes thirteen cases in which with more or less difficulty the site of obstruction was reached through the inguinal canal (at the time the paper was written hardly anyone thought of performing median laparotomy), but sixteen cases are contrasted with these in which the operator could neither define nor relieve the obstruction by this route.

In cases where symptoms of intestinal obstruction co-exist with a history of recent hernia, the patient's safety will generally depend upon the prompt performance of a median abdominal exploration.

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Other references are given in Mr. Makins' paper, and various ones by Krönlein.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. xi, p. 97.)

THE CLINICAL EXAMINATION OF THE HÆMIC LEUCOCYTES

BY

ARTHUR G. PHEAR, M.D.

I. Introductory.

THE object of this communication is to draw attention to the advantages of fluid preparations over the cover-slip method more commonly in vogue in the study of the white corpuscles of blood. In the cover-slip method a thin film of blood is spread on the cover-slip, fixed, and subsequently stained. An initial difficulty is the preparation of a film in which the corpuscles are evenly arranged, so that the cells may be individually observed; the relatively scanty leucocytes are apt to be obscured by the overwhelming numbers of the red corpuscles.

Then, however carefully the film is prepared, there is inevitably some flattening of the cells with distortion of outline, and the preparation is thereby rendered useless for the purpose of accurate drawing or measurement. Further, in making the differential count, there is the uncertainty as to whether the leucocytes are fixed on the cover-slip in the proportion in which they exist in the blood ; some may be destroyed in the process of making the film ; some may be washed away ; others are obscured by the red corpuscles; the outcome in practice is that counts of different films taken from the same blood often give materially different results.

These difficulties are, for the most part, met by examining the blood as a fluid preparation, having previously diluted it with a suitable fluid medium. A fluid should be used of a kind to preserve the white cells as far as possible without distortion, and to this a stain should be added of a strength to bring out clearly the structural detail of the cell without too far modifying its translucency. The thickness of the unflattened cell treated in this manner is an advantage, since, by gradually focussing from above downwards through the depth of the cell, views are obtained at different focal planes which are equivalent to a series of horizontal sections, and a good idea is gained of the true shape and disposition of the nucleus and other structures within the cell.

In one respect the cover-slip film is preferable to the fluid preparation. The fixed film of blood can be mounted and preserved as a permanent object, whereas the fluid preparation is in good condition only for a limited time, beginning to deteriorate in the course of a few days. In clinical work, however, all that is usually desired is to ascertain and record the numerical proportions of the several kinds of leucocyte ; no permanent preparation is needed. It is only when unusual forms of leucocytes are present that permanent specimens are required, and in this event they can readily be made, though in every

case it is desirable to supplement the permanent film by camerâ lucidâ drawings of the undistorted corpuscles, as they appear in a fresh fluid preparation.

Of the innumerable stains and solvents that are available, I find that the best for general use is a ·2 per cent. solution of methylene blue (Grübler) in alcohol 40 per cent.[1] In this solution the distinctive features of the leucocytes are preserved, while the red corpuscles lose their colour and become relatively inconspicuous.

II. METHOD.

(*a*) A small quantity of the methylene blue solution is drawn up into a pipette. The pipette for the enumeration of the white blood corpuscles supplied with the Thoma-Zeiss apparatus is convenient for this purpose. The fluid should be drawn up to the mark 0·5 on the pipette, since this quantity forms a film of convenient depth when the operation is completed.

(*b*) The finger, having been carefully cleaned, is pricked with a sharp needle, and a fairly large drop of blood is allowed to exude.

(*c*) A glass slide is brought into contact with the drop of blood, and as much blood removed as adheres to the slide. Care should be taken to keep the glass slide from touching the finger.

(*d*) The methylene blue solution is now slowly blown out from the pipette on to the drop of blood, and the two are allowed to mingle. When the fluid has all been expelled, the end of the pipette is wiped, and a current of air is directed through the pipette *on to the surface* of the drop which lies on the slide. By this means eddies are set up, and in a few seconds the blood and methylene blue solution are thoroughly and uniformly mixed ; as

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[1] A dilute solution of methylene blue in 40 per cent. alcohol, to which a trace of caustic potash and of osmic acid is added, is recommended by Kanthack and Hardy, who found it especially valuable for the study of the finely granular basophile cell. See ' Journal of Physiology,' vol. xvii, p. 88 ("Wandering Cells of Mammalia ").

they mingle the colour of the fluid changes from blue to a deep green.

(e) A cover-glass, seven eighths of an inch square, is placed over the fluid, and the latter spreads out as a thin film, none of which exudes beyond the margin of the cover-slip if the above directions have been closely followed. The edges are then sealed with vaseline, and a preparation is obtained which may be examined at leisure with an immersion lens. The whole process takes about three minutes.

In working with a high power, it is important to regulate the light, as many details of the cell structure are seen to best advantage with a comparatively dim light. The diaphragm should be adjusted until the most favourable illumination is obtained.

On examination of the fluid film, it is found that the red corpuscles have lost their colour, and are visible in outline only as transparent discs, which do not interfere with the study of the white corpuscles. The white corpuscles are therefore the only conspicuous objects in the field, and of these the several varieties can readily be distinguished, notwithstanding the absence of an acid stain. That the cells are free from the distorting influence of pressure may be shown by measuring the distance from the upper surface to the lower surface of the cell by means of the fine adjustment. This can readily be done in the case of the coarsely granular cell, of which both the upper and the lower limits can be easily and clearly defined. The diameter from above downwards, in every case that I have measured, corresponds almost exactly with the diameter from side to side; the cell is therefore approximately spherical.

The rapidity with which the fluid film can be prepared and the uniformity of the result render the method well suited for clinical work. The aspect of the leucocytes is, however, in certain respects, peculiar and different from their appearance in a dried film made in the more ordinary way, and I therefore ask leave to add a short

description of the leucocytes as they appear in a fluid film made from normal blood, as a preliminary to the employment of the method in the study of forms that may deviate from the normal.

III. THE NORMAL LEUCOCYTES.

Five kinds can be distinguished in such a preparation. In order of frequency, these are :—

(1) Rounded cells, with polymorphous nuclei and finely granular protoplasm (*"finely granular oxyphile cells"* or *" polymorphonuclear cells"*).

(2) Rounded cells, with single rounded nuclei and clear protoplasm which stains feebly or not at all with methylene blue. The group includes the *lymphocyte*, the *large hyaline cell*, and intermediate grades.

(3) Large cells, usually oval, with single nuclei of irregular shape, and protoplasm which stains diffusely with methylene blue.

(4) Rounded cells, with protoplasm containing large refractile granules (*" coarsely granular oxyphile cells,"* or *" eosinophile cells"*).

(5) Small cells, usually rounded, with lobed nuclei, and protoplasm which stains a diffuse mauve or purple tint with methylene blue, and contains granules of medium size, and deep blue or violet colour. The granules are often aggregated at one or more points peripherally (*"finely granular basophile cells"*).

The Polymorphonuclear Cell.

Contour, rounded.

Diameter,[1] 9 to 10 μ.

Numerical proportion, 45 to 60 per cent.

The *protoplasm* of this cell does not stain with methylene blue, and shows no structure beyond a fine granula-

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[1] Measurements were taken of 100 cells. The diameter was 8·5 μ in 2, 9 μ in 16, 9·5 μ in 41, 10 μ in 38, 10·5 μ in 2 cells, and 11 μ in 1 cell.

tion. The granules are minute, numerous, and evenly diffused throughout the protoplasm. When in focus they appear as dark dots. They have no affinity for methylene blue, being no more conspicuous in an alcoholic solution of methylene blue than in an alcoholic solution of similar strength in which no methylene blue is dissolved.

The *nucleus* is a complex body consisting of several rounded lobes, disposed irregularly in the central part of the cell. At first sight the lobes may seem to be separate from one another. If, however, the focus be slowly changed, it can, in almost every instance, be made out that connecting strands, of varying thickness, pass from one lobe to another. There is, in fact, no break in the continuity of the parts of the nucleus, which exists as an elongated body, much contorted, in places deeply constricted, elsewhere bulged into rounded swellings, of which there are three, four, or five, rarely a greater number. As the focus is altered, one part after another of the nucleus comes into view, and it is appreciated as a structure of substantial thickness, the limits of which can be defined in the vertical as well as in the horizontal plane (see Fig. 1).

FIG. 1.
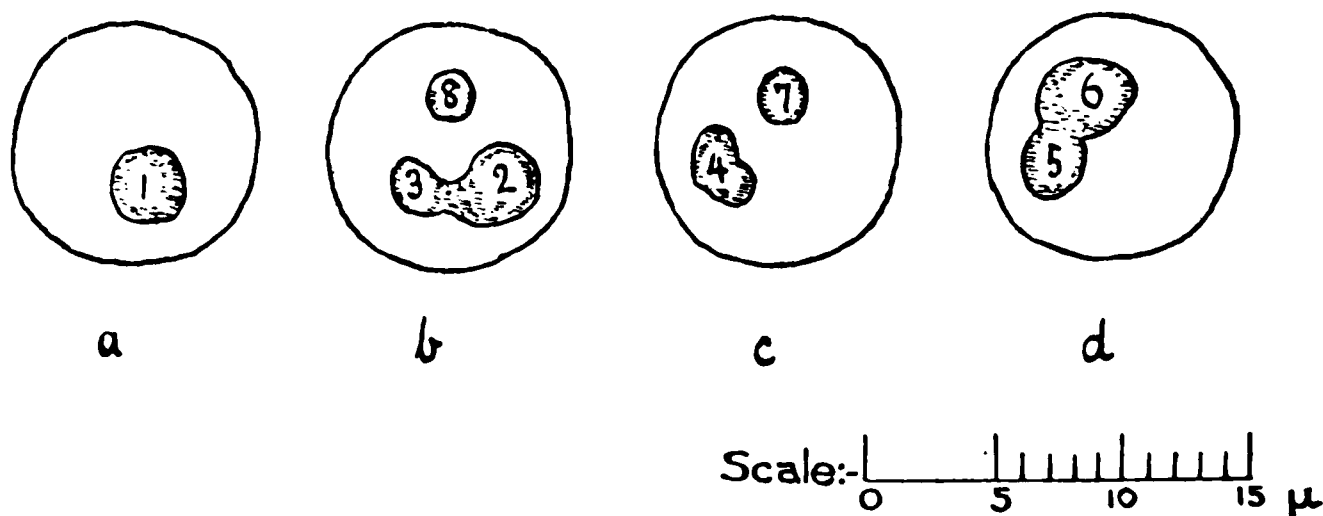
Drawings of a polymorphonuclear cell at different focal planes, to show the continuity of the parts of the nucleus. The letters *a* to *d* indicate the focal planes from above downwards. The loose coil of the elongated nucleus can be traced by following the numbers 1 to 8.

The cells represented in this and subsequent figures were all drawn in outline with camera lucida.

Occasionally the nucleus is S-shaped, or arranged in the form of a horseshoe, and lies in one plane; it is then obviously an undivided body. Less commonly the parts of the nucleus are closely coiled and form a rounded mass resembling the simple rounded nucleus of the hyaline cell; but on close observation the intricacy of its structure has always been evident; I have never observed in my preparations, in which the death of the corpuscle is almost instantaneous, the rounded nucleus described by Sherrington [1] in cells which he allows to die slowly.

The nucleus is coloured deeply with methylene blue, though not so deeply as in the case of the hyaline leucocytes. One or more points, more strongly stained than the surrounding parts, indicate the position of nucleoli.

The group of polymorphonuclear cells is sharply defined; no cells are present in normal blood intermediate in structure between the cells of this and other groups of leucocytes.

Rounded Cells, with single rounded Nuclei and clear Protoplasm.

The group includes :—

(a) **Lymphocytes,** in which the nucleus, occupying nearly the whole cell, is surrounded by only a thin envelope of protoplasm.

(b) **Large hyaline cells,** in which the diameter of the cell is at least twice the diameter of the nucleus.

(c) **Intermediate forms.**

Diameter, varies from 6 to 10 μ, rarely more.

Numerical proportion, 35 to 45 per cent. of total number of leucocytes.
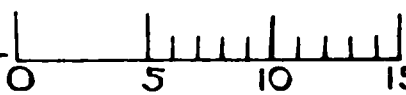
A prominent feature of this group is the great variability in the size of the cells which compose it. All gradations between the lymphocyte and the large hyaline cell are present and readily found in normal blood, and

[1] 'Proc. Internat. Congress of Physiologists,' Liège, 1892.

it is impossible to split the series into any but the most arbitrary divisions, since the members of the group resemble one another in every essential point except that of size. The cells of intermediate size are probably rightly regarded as transitional forms, or as stages in the growth of the lymphocyte into the larger hyaline cell. The size of the nucleus is remarkably constant throughout the series; its diameter is seldom less than 4·5 μ or more than 5·5 μ. The growth of the cell is, therefore, chiefly an increase of the extra-nuclear protoplasm.

FIG. 2.
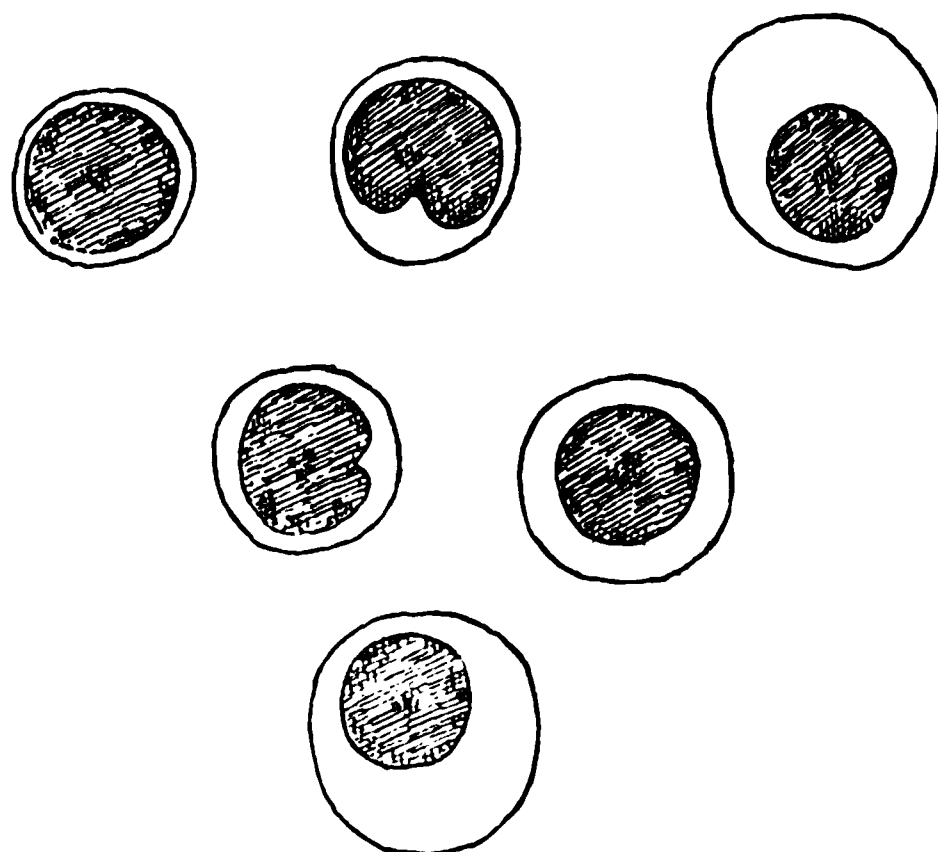
Cells showing gradations from the lymphocyte to the large hyaline cell.

The larger cells of the group correspond with the "myelocytes" of Sherrington.[1]

The *protoplasm* of these cells is always clear, free from granules, and remains in the methylene blue solution for some time without taking the stain, or at most being faintly tinged with blue. Eventually an exceedingly fine

[1] *Vide* 'Proceedings of the Royal Society,' vol. lv, p. 193. The name "myelocyte" is more commonly given to certain cells that are not represented in normal blood.

granulation becomes visible, probably due to staining of the nodes of the protoplasmic network.   There is never a strong diffused blue coloration of the protoplasm, as in certain cells to be described.

The *nucleus* is a simple, undivided structure, with a round outline, which in many cases is broken by an indentation, more or less deep.   The notch is more often present than at first sight appears; for it is only conspicuous when in profile, *i. e.* when the position of the cell is such that the notch is situated laterally.   If, from the position of the cell, the notch lies, not at one side, but above or below, the visible outline of the nucleus is not broken by the notch.   Occasionally in the fluid preparation a lymphocyte, whose nucleus appears round, rolls over, and with the change of position an indentation comes into view.   The nucleus stains with methylene blue more strongly in this than in any other kind of leucocyte.   All parts of the nucleus do not, however, take the stain equally well; the colour is concentrated at certain points and areas within the nucleus, and the result is a patchy or mottled appearance.

Large Oval Cells, with irregular Nuclei, and Protoplasm which stains diffusely with Methylene Blue.

The cells about to be described are not usually recognised as forming a distinct group of the hæmic leucocytes.   They are probably classed in most accounts with the large hyaline cells, though in several respects they differ remarkably from the large hyaline cell as it is commonly figured.   In the plate accompanying the communication on " Wandering Cells of Mammalia," by Kanthack and Hardy, the large hyaline cell appears as a rounded body, with rounded nucleus, the latter being deeply stained with methylene blue, while the cell protoplasm is not stained.   In the case of the cell to which attention is now drawn, the contour of neither cell nor

nucleus is round, and the protoplasm is readily stained in solutions of methylene blue.

The cells are remarkable both for their size and shape ; they are as a rule oval, and larger than the leucocytes of any other group ; in length they vary from 10 to 14 μ, and in breadth from 7 to 10 μ. They are always to be found in normal blood, and their proportion to the total number of leucocytes in circulation is fairly constant, ranging from 5 to 10 per cent.

FIG. 3.

Scale:-

Large oval cells, showing various forms of the irregular nuclei.
The faint shading indicates the staining of the protoplasm.

The *protoplasm* is peculiar in its affinity for methylene blue ; it takes the stain rapidly, becoming coloured a uniform and rather opaque blue, without at first a trace of granulation. The coloration is visible in the most recently prepared specimen, and is not subsequently deepened to any extent, though a fine granulation may become evident, perhaps due to the more intense staining of the nodes of the protoplasmic network. The colour reaction with methylene blue is characteristic ; in the polymorphonuclear cell the finely granular protoplasm remains unstained ; in the lymphocyte and larger hyaline cells the staining of the protoplasm is comparatively faint, and is not evenly diffused, but appears as a fine blue granulation ; in the coarsely granular leucocyte the staining of the large granules gives to the cell, as a whole,

a greenish tint, while the protoplasm of the " basophile " cell acquires a mauve or purple tint, not likely to be confounded with the opaque blue coloration of the large oval cell under notice.

The *nucleus*, always irregular, varies much in shape. It is large, occupying a considerable part of the cell, and is usually elongated in the direction of the long axis of the cell. The form commonly seen is that in which the outline on one side forms an unbroken curve, while on the other side it is interrupted by one, two, or three notches, giving to the whole structure a lobed or reniform shape. Sometimes there appear to be two separate oval nuclei, lying obliquely across the cell, one at either end; as the focus is lowered, a broad connection comes into view between the two lobes, which are, in fact, the bulged extremities of a single reniform nucleus, lying with the notch upwards (see Fig. 4). The lobes of the nucleus are occasionally arranged in an irregular cup-like manner around a central depression. Other forms are drawn in outline (Fig. 3), from which it is seen that the general

FIG. 4.
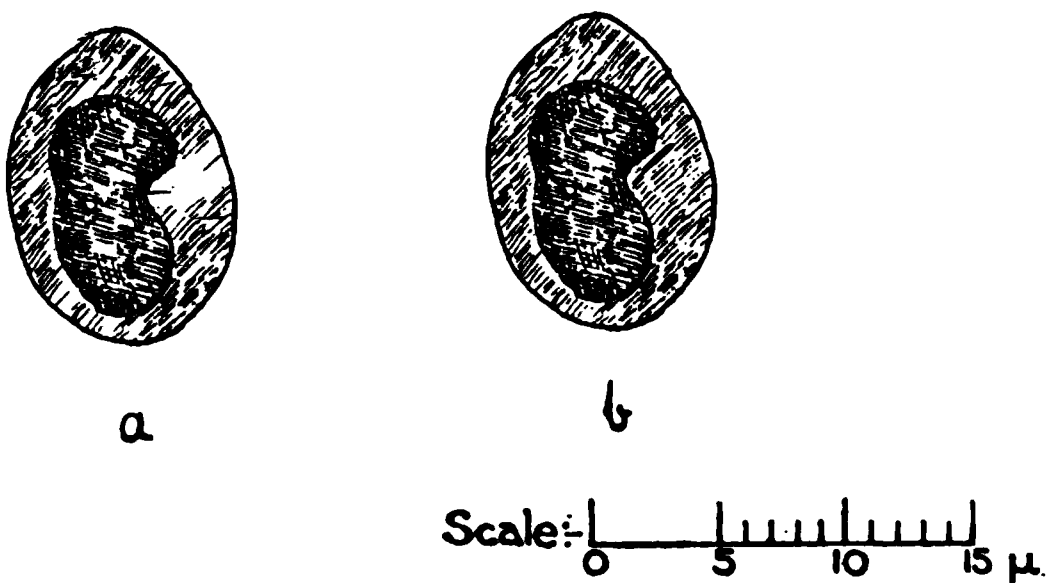
Large oval cell. The same cell is drawn at two focal planes. At the upper plane (*a*) there appear to be two nuclei. On lowering the focus these are seen to be the extremities of a large single nucleus.

characters of the nucleus are constant, though in detail of outline it exhibits much diversity.

The nucleus stains with methylene blue, but not so

strongly as in the preceding. groups.   Two or more nucleoli can usually be distinguished.

Transitional Forms between the Lymphocyte and the Cell just described.

Cells intermediate in structure between the lymphocyte and the large oval cell are found circulating in the blood-stream, and, indeed, are so far from uncommon as to give reason for supposing that a certain number of the lympho-cytes develop while in circulation into the larger and more differentiated cells.   The transitional forms include cells which resemble lymphocytes in size and every other respect except that the nucleus, which nearly fills the cell, is not rounded, but elongated, and sharply bent on itself in the form of a half spiral.   One of these cells is figured (Fig. 5), the coiled arrangement of the nucleus being shown by camerâ lucidâ drawings taken at different

FIG. 5.
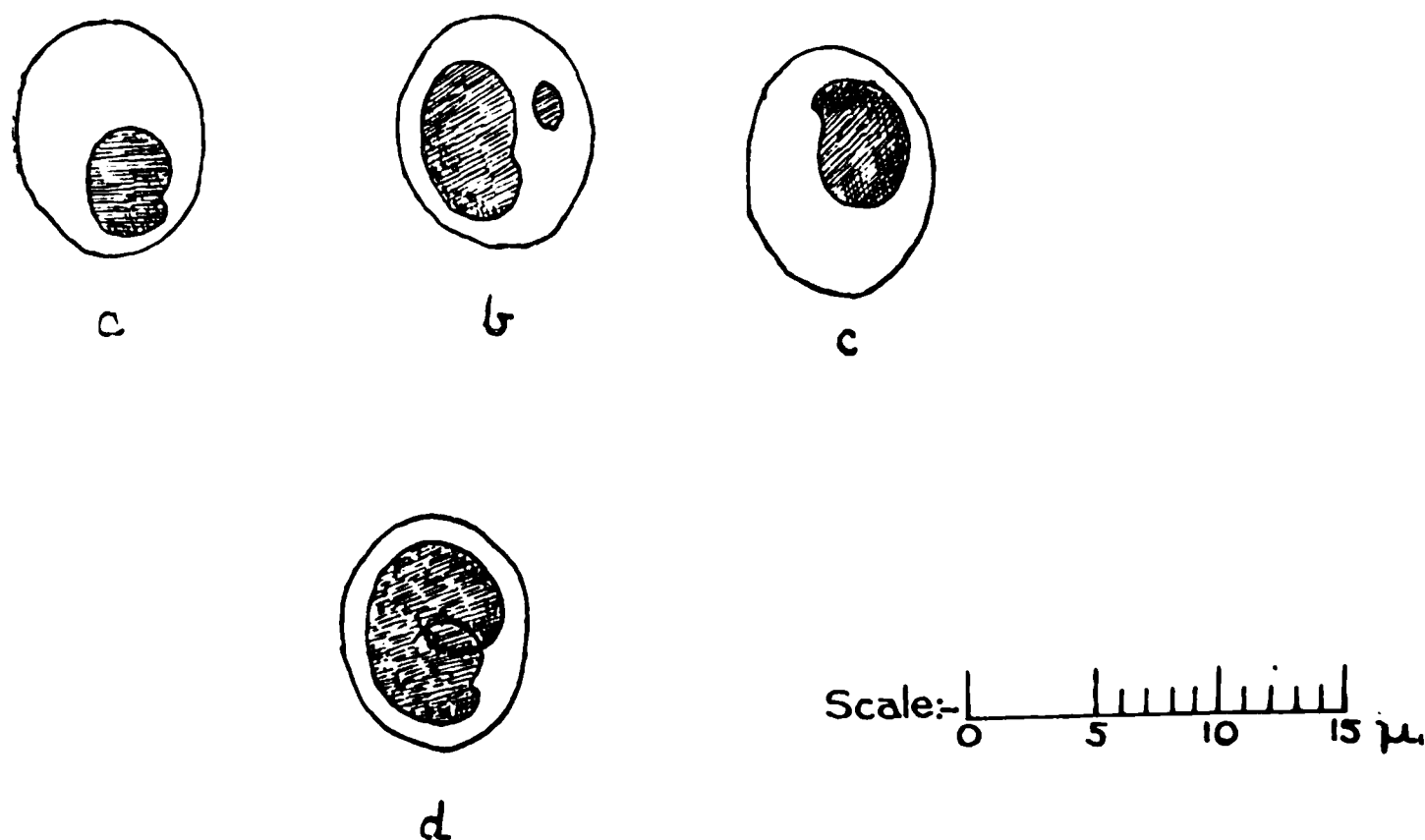
a, b, and c are drawings of one cell at three focal planes, showing the closely coiled nucleus.   In d the parts of the nucleus are shown superimposed.

focal planes.   In other cells, no larger than lymphocytes, the nucleus has a cup-like form, the nuclear indentation

(see p. 339) being deepened, with thickened and irregular margins. Other cells may be found, larger than the lymphocyte, in which there is a broader margin of protoplasm, and in which the protoplasm stains diffusely with methylene blue, and surrounds a nucleus of irregular form.

The lymphocyte is the only cell to which the large oval cell has obviously close relations; no transitional forms are found between the large oval cell and any other kind of leucocyte in normal blood.

It has already been pointed out that the lymphocyte is closely related to the large hyaline cell. From the study of the leucocytes in circulation, it would therefore appear that the lymphocyte is an immature form which may develop either into the large hyaline cell with rounded nucleus and clear protoplasm, or into the larger oval cell with irregular nucleus and protoplasm which stains opaquely and diffusely with methylene blue. Moreover, it is probable that the growth of the lymphocyte into either of these two forms occurs while it is actually in circulation, since the intermediate stages of its growth are commonly present and readily found in the blood-stream. It may be remarked here that, so far as the study of the hæmic leucocytes goes, there is no ground for supposing that the lymphocyte develops into any form of leucocyte other than the two mentioned. Intermediate forms are not present in normal blood between the lymphocyte on the one hand and the polymorphonuclear cell, the coarsely granular cell, or the " basophile " cell on the other.

The Coarsely Granular Cell.

Contour, rounded.

Diameter, 9·5 to 10·5 μ.[1]

Numerical proportion, 1 to 2·5 per cent.

[1] Measurements were taken of 50 cells. The diameter was 9 μ in one cell, 9·5 μ in 8, 10 μ in 35, 10·5 μ in 5 cells, and 11 μ in one cell. The diameter usually given is too large, perhaps because the measurements are taken from dried films, in which the cells are necessarily flattened.

FIG. 6.
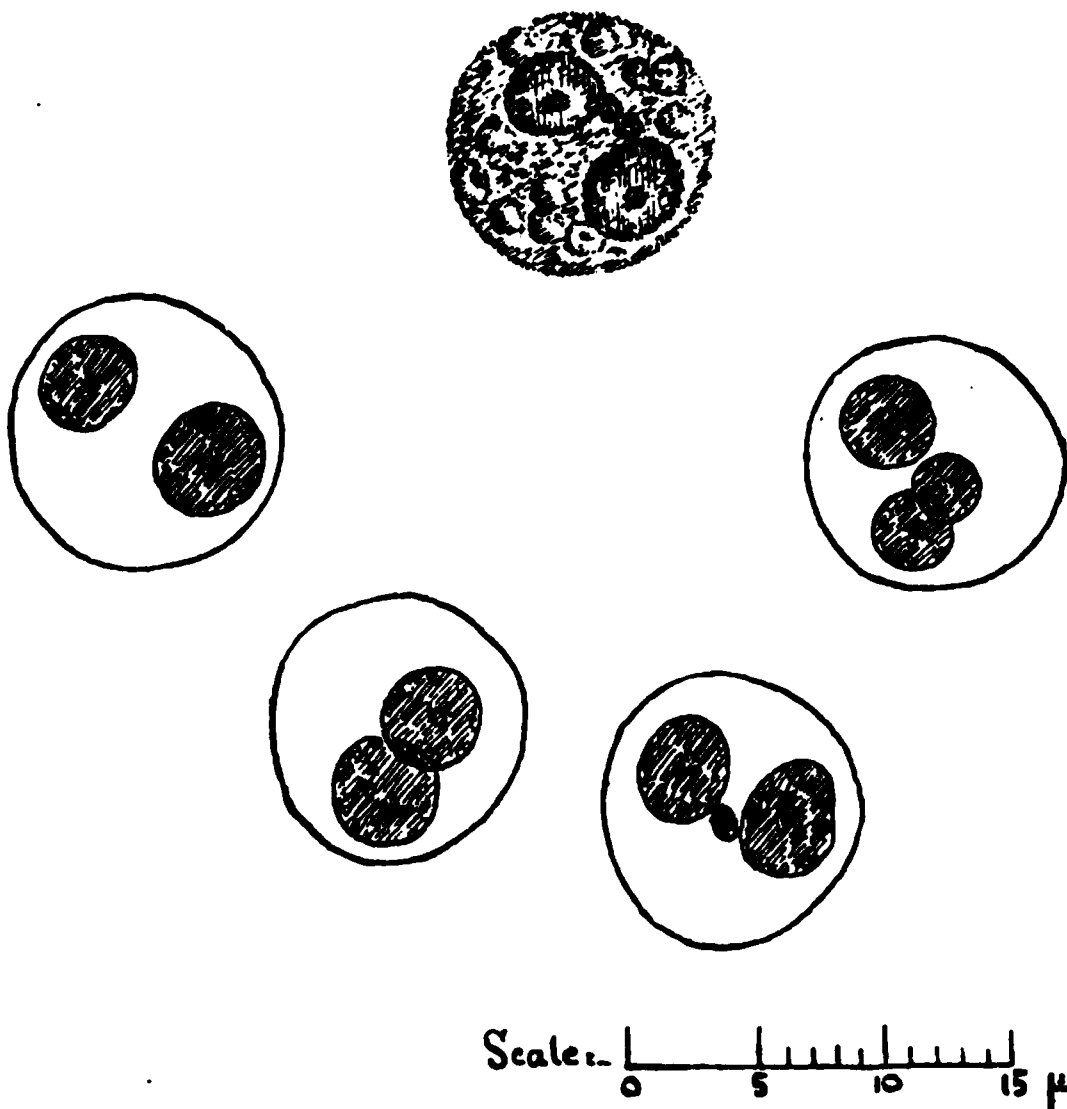
Drawings of coarsely granular cells (the lower ones in outline only),
showing various forms of nuclei.

In fluid preparations made with methylene blue the
attention is immediately arrested by the coarsely granular
cells, not only on account of the large refractile granules.
which they contain, but by reason of their colour.

The *granules* have some affinity for methylene blue,
and in the fluid preparations are stained, not blue, but a
peculiar green or yellowish-green colour, which gives to
the whole cell a striking appearance. The individual
granule, when its upper surface is in focus, appears as
a glistening object, rounded or slightly oval, with dark
outline ; as the focus is lowered the glistening appearance
is lost, and at the focus of its equatorial plane the granule
is seen as an opaque body of a uniformly dark green,.
almost black colour.

The *nucleus*, often placed excentrically, is not so com-
plex as that of the polymorphonuclear cell ; it is usually

bi-partite, each part being rounded, and containing a nucleolus. A connecting strand between the two parts can sometimes be distinguished; often it is obscured by the granules. Less commonly the nucleus is tri-partite, or assumes other forms, some of which are figured. The deep blue stain of the nucleus is in marked contrast to the greenish colour of the rest of the cell.

The " Basophile " Cell.

Contour, rounded, rarely oval.

Diameter, 8 μ.[1]

Numerical proportion, ·3 to 1 per cent.

Fig. 7.
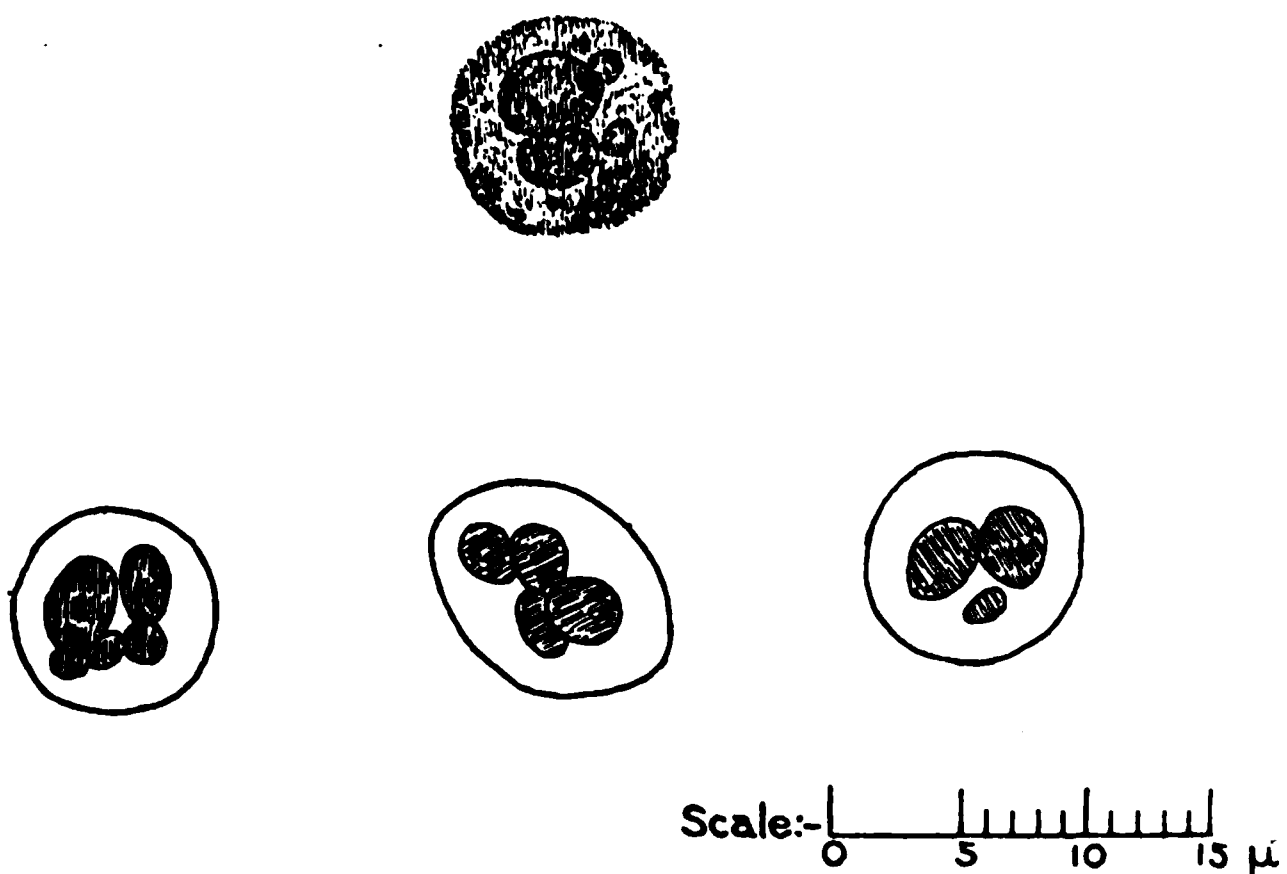
" Basophile " cells, showing lobulation of nucleus. In the detailed drawing the peripheral aggregation of the granules is indicated.

The basophile cell is among the smallest and is the rarest of the leucocytes present in normal blood. In

[1] Measurements were taken of 29 cells. Of these the diameter was 7 μ in one cell, 7·5 μ in 2, 8 μ in 15, 8·5 μ in 4, and 9 μ in 3 cells. Four cells were oval; two of these measured 7 μ × 8 μ; the remaining two were 7 μ × 9 μ

outline it is usually round, exceptionally oval. The *protoplasm* stains diffusely with methylene blue, the resulting tint being not a pure blue, but a mauve or purple. This colour reaction is distinctive, though the cause of it is not clear ; it is probable that the basophile cell has a special affinity for methylene red, which is often present in small quantity in specimens of methylene blue. The protoplasm contains granules, usually described as fine, but in human blood, at any rate, intermediate in size, being coarser than the fine granules of the polymorphonuclear cell, and finer than the large granules of the coarsely granular cells. The granules stain deeply with methylene blue, and are apt to be massed peripherally in one or more dark violet clumps, close to the surface of the cell.

The *nucleus* is a complex structure, consisting of rounded lobes, of which there are seldom less than four, often five, connected with one another, and usually aggregated in a compact mass near the centre of the cell. The parts of the nucleus are stained a dull slate or grey colour with methylene blue.

The above account of the leucocytes is based on preparations made with a ·2 per cent. solution of methylene blue in alcohol 40 per cent. Other stains and other media may be used in the preparation of the fluid film without sacrificing the advantages of the fluid method, and although I believe an alcoholic solution of methylene blue to be the most useful medium for clinical work, yet for special purposes, such as the investigation of the staining affinities of certain granules, it is necessary to vary the nature of the stain, and sometimes of the fluid used for dilution.

In **alcohol 40 per cent.** (without stain) the red corpuscles are laked. The *polymorphonuclear leucocytes* show much detail of structure. The fine granules appear as small, distinct, dark points ; they are as conspicuous as in a methylene blue preparation. The outline of the complex

nucleus is sharp, and the nuclear network is often beauti-
fully clear, more so than in most stained specimens. The
nuclei of the *lymphocytes* and *larger hyaline cells* are also
remarkable for their sharp outline with distinct nuclear
reticulum and nucleolus. The protoplasm of these cells is
clear and free from granules. The protoplasm of the *large
oval cells* displays an immense number of dark granular
points, very fine, a few about the same size as, but the
larger number finer than, the granules of the polymorpho-
nuclear cell. The *coarsely granular corpuscles* are striking
objects. The large granules, owing to their refractile
nature, appear as glistening bodies when their upper sur-
face is in focus, changing to rounded black dots as the
focus is lowered to that of the equatorial plane.

In a solution of **osmic acid 1 per cent.** (without stain) the
red corpuscles are not laked. The fine granules of the
polymorphonuclear cells are visible as dark dots, and are
more sharply defined than in the alcohol 40 per cent.
preparation. The position of the nucleus is indicated by
a clear space, free from granules ; there is no distinct
nuclear outline nor reticulum. In many of the *lymphocytes*
a few dark granules of large size are visible in the proto-
plasmic rim. I have not seen these in any but osmic
acid preparations. The granules of the *coarsely granular
cells* are conspicuous objects, but appear no darker in
osmic acid than in alcohol preparations.

Osmic acid (1 per cent.) fixes the *red corpuscles* with-
out distortion, and it is a convenient solution with which
to prepare fluid films when red corpuscles of abnormal shape
are present. Only a minute drop of blood should be
taken, and this should be thoroughly mixed with the
diluting solution, to which a small quantity (·2 per cent.)
of methyl green may with advantage be added.

Acetic acid (·3 per cent., as recommended by Thoma)
causes the red corpuscles to disappear, but is not a good
medium in which to examine leucocytes, since it rapidly
produces changes in their structure. In a solution of
methylene blue (·2 per cent) in acetic acid (·3 per cent.)

the structure of the *polymorphonuclear cells* is for a few minutes beautifully distinct; the cell then becomes, as it were, ballooned on one side, a crescentic space appearing between the main mass of the cell and an external envelope, which seems to have separated from the granular protoplasm. In many cells the change goes no further for a considerable time; in a few the cell-protoplasm quickly becomes swollen and ill-defined, the nucleus only retaining its form and stain; ultimately the nucleus swells, becomes misshapen, and its colour fades. Similar changes take place in some of the *lymphocytes*, a thin outer layer separating and standing away from the body of the cell. Lymphocytes that are swollen in this way acquire a likeness to the large hyaline cells, and are liable to be estimated as such in the course of a differential count. Of *coarsely granular cells* only a small number is found, and these are materially altered. The cell, as a whole, is swollen; the granules are not visible as discreet bodies, but are broken down into a homogeneous transparent substance of a yellowish-green colour; the nucleus is ill-defined and feebly stained. In differential counts made with acetic acid preparations, the percentage of coarsely granular cells is considerably below the average, and probably many of them are actually destroyed.

Eosin in alcohol (40 per cent.).—The appearance of the *coarsely granular cell* in fluid preparations made with a dilute solution of eosin is very remarkable. A solution of convenient strength may be obtained by drawing a saturated solution of eosin in 40 per cent. alcohol up to the mark 1 on the white corpuscle pipette (Thoma-Zeiss), the bulb is then filled to the mark 11 with alcohol 40 per cent. A dilution of 1 in 11 is thus obtained, and with this fluid the preparation is made in the ordinary way. The result is striking in normal blood, and still more so when there is an excess of coarsely granular cells. The large granules stand out as conspicuous objects, highly refractile and coloured a deep crimson, while the other leucocytes are none of them more than feebly stained.

It is noteworthy that the so-called "fine oxyphile granules" of the *polymorphonuclear cells* are not stained at all deeply in this solution.   The red corpuscles are laked.

Methyl green (·4 per cent.) in alcohol (40 per cent.).[1]—The red corpuscles are laked.   The *nuclei* of all the leucocytes take the stain brilliantly ; the protoplasm in all cases remains unstained.   Methyl green is one of the most satisfactory stains for the nuclei of the lymphocytes and the larger hyaline cells ; they stain sharply and clearly with but little loss of translucency.   It is remarkable that the *protoplasm* of the large oval cell and of the " basophile cell," both of which stain characteristically with methylene blue, is unstained in solutions of methyl green.

The small *granules* of the polymorphonuclear cell have no affinity for methyl green.   The granules of the basophile cell are not stained, notwithstanding the fact that methyl green is a double salt in which the colouring principle plays the part of a base.   The large granules of the coarsely granular cell are tinged with green.

IV. The Differential Count.

For making a differential count of the leucocytes the fluid film prepared with an alcoholic solution of methylene blue is convenient, since in this the red discs are inconspicuous, and the white corpuscles, which it is desired to enumerate, alone attract the attention.   The method also appears to fulfil the conditions necessary to an accurate enumeration.   It is essential to accuracy that the blood should be uniformly mixed with the diluting fluid.   That this is so in the fluid preparation, I have satisfied myself by making separate counts over different areas of the same preparation ; the results have always been close, the variation in the case of the commoner forms amounting to no more than 2 or 3 per cent.   I find also that different preparations made from the same individual, at

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[1] The solution rapidly undergoes changes and therefore must be freshly made.

the same time, tally closely in the relative proportion of the several leucocytes; from this uniformity it may be inferred that the percentage of the leucocytes is substantially the same in the fluid film as in the blood, and that the preparation is a reliable index to the state of the blood.

The greater the number of leucocytes brought under notice, the more accurate are the results arrived at. No fewer than 500 should be counted. A mechanical stage is almost a necessity ; it not only ensures that all the leucocytes within a given area are included in the count, but it further prevents the risk of going over the same ground twice. It has been my practice to count the cells with an immersion lens ; the extra time involved is well spent, since it is seldom that any uncertainty is felt as to which group a given leucocyte should be placed in when a one-twelfth immersion lens is used ; whereas, in working with a lower power, this difficulty not infrequently arises.

With a view of testing the somewhat varying statements on the relative proportions of the several kinds of leucocytes in healthy human blood, I have made a series of forty-nine observations, in the course of which a total of over 26,600 leucocytes were counted. Dealing with these figures as a whole, the percentages were as follows :

Polymorphonuclear cells, 54.

Rounded hyaline cells, 36 (of these 18 per cent. were lymphocytes).

Large oval cells, 8·4.

Coarsely granular cells, 1·9.

Cells with basophile granules, ·7.

For each kind of leucocyte a further calculation was made, the mean being taken, in the first place, of the percentages which were in excess of the average for that leucocyte, and, in the second place, of the percentages which were below the average. In this way what may be termed a mean maximum and a mean minimum percentage has been estimated for each leucocyte.

TABLE I.

Total number of leucocytes counted, 26,644.
Number of observations, 49.

	Mean minimum percentage.	Mean maximum percentage.
Polymorphonuclear cells . . .	48	59
Rounded hyaline cells . . .	30	40
Large oval cells	6·8	11
Coarsely granular cells . . .	1·2	2·5
" Basophile " cells	·4	1·1

In estimating the percentage of the various leucocytes, some authorities take no account of the lymphocytes on the ground that they are immature forms. There is, however, great difficulty in deciding which corpuscles are immature and which have reached their full development. It is likely that, of the group of cells with hyaline protoplasm, all but the largest hyaline cells are as properly termed immature as the lymphocytes, and we have no certain knowledge, even with regard to the largest hyaline cells, as to whether they have attained the final stage of their growth. I have, therefore, preferred not to discriminate between adult and immature forms, but to include in the count all the leucocytes that were seen.

The observations from which the figures in Table I are computed were made on more than one individual, and not all at the same hour of the day. Among them is included a series of observations, made on one individual and at the same hour of the day, the time chosen being that at which the influence of meals is least likely to be felt, viz. immediately before breakfast. In this series 6000 leucocytes were counted in the course of ten observations, and the results are summarised in Table II.

TABLE II.

Total number of leucocytes counted, 6000.

Number of observations, 10; made on one individual, on each occasion before the first meal of the day.

	Smallest percentage observed.	Mean minimum percentage.	Average percentage.	Mean maximum percentage.	Largest percentage observed.
Polymorphonuclear cells . . .	47	49	50	53	54
Rounded hyaline cells . . .	37	38	40	42	45
Large oval cells . . .	4·5	5·7	6·9	8	10
Coarsely granular cells . . .	·8	1·5	2	2·6	3·4
"Basophile" cells . . .	·2	·3	·45	·6	·8

It is interesting to note the small range within which the percentages of the two common forms of leucocyte varied —in the case of the polymorphonuclear cell between a mean minimum of 49 per cent. and a mean maximum of 53 per cent., and in that of the rounded hyaline cells between 38 and 42 per cent. The range of variation is naturally much greater proportionately for the leucocytes of rare occurrence than for the commoner forms. In the case of the coarsely granular corpuscle and the basophile corpuscle, the numbers that actually passed under observation in the course of the second series of ten counts were so small as to afford no secure basis for the calculation of averages. I prefer, therefore, to rely on the larger numbers dealt with in Table I, as indicating more accurately the average occurrence of these rarer leucocytes.

Further observations that I have made all tend to show that for one individual the daily variation in the relative proportions of the leucocytes is not great, provided the disturbance occasioned by meals be avoided. Thus in one case the percentage of the polymorphonuclear

cells on four consecutive days was 47, 48, 46, and 44; the percentage of the hyaline cells was 42, 40, 42, and 43, including lymphocytes, the numbers of which were 14, 10, 12, and 12 per cent.; large oval cells were present in the proportion of 9, 11, 9, and 9 per cent.

I believe it to be an essential condition that a long interval be allowed to elapse between the last meal and the occasion of making the count. The most convenient time is doubtless before breakfast; at no other hour of the day can the disturbing influence of meals be eliminated, and for no other time is a normal standard available for comparison.

For a healthy individual the following figures may be taken as representing the limits within which the percentage of the several cells usually lies at the time stated; it is not to be inferred that transgression of these limits is necessarily of pathological import, only that under circumstances of health it is exceptional for the limits to be exceeded in either direction.

Polymorphonuclear cells 45 to 60 per cent.

Rounded hyaline cells, including lymphocytes, large hyaline cells, and intermediate forms, 35 to 45 per. cent.

Lymphocytes, in which nearly the whole cell is occupied by the nucleus, constitute from 10 to 20 per cent. of the total number of leucocytes.

Large hyaline cells, arbitrarily defined as those in which the diameter of the whole cell is at least twice the diameter of the nucleus, are not present in greater numbers than 2 per cent.

Large oval cells 5 to 10 per cent.

Coarsely granular cells 1 to 2·5 per cent.

Cells with basophile granules ·3 to 1 per cent.

The percentage for the polymorphonuclear cells (45 to 60 per cent.) is considerably below that stated by most authorities. Copeman[1] gives 75, Schäfer[2] 70 to 90, and

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[1] 'System of Medicine,' Allbutt (1898), vol. v, p. 416.

[2] 'Text-book of Physiology' (1898), p. 152.

Kanthack and Hardy[1] 75 to 90, as the normal percentage in human blood. The latter authors excluded "immature" cells from their count; this partly explains the discrepancy between their results and mine. Also the figures given by me are based on observations made at the hour when the proportion of the polymorphonuclear cells is at its lowest. There is a fairly constant decrease of about 10 per cent. between 10 o'clock at night (*i. e.* about two hours after dinner) and 8.30 the next morning, and at no other time of day is the percentage of these cells so low as in the early morning before breakfast. I do not, however, propose to deal with the diurnal variation of the hæmic leucocytes, either as regards their total number or their relative proportions. The problem relating to the part played by meals and other factors in influencing their variation is an intricate one, and I have already occupied too much time in the discussion of matters that may seem to be more of physiological than of pathological interest; yet I must claim that from the clinical standpoint they are of importance, and on this ground I ask your indulgence.

[1] Loc. cit., p. 92.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. xi, p. 109.)

DESCRIPTION OF PLATE VIII

Illustrating the Clinical Examination of the Hæmic Leucocytes (ARTHUR G. PHEAR, M.D.).

The leucocytes are shown as they appear in fluid films prepared with the methylene blue solution described in the text.

The reaction of the several cells with methylene blue is indicated. The protoplasm of the polymorphonuclear cell (*a*) and of the lymphocyte (*b*) is unstained; that of the large hyaline cell (*c*) shows a faint blue granulation; the protoplasm of the large oval cell (*d*) is stained a diffuse blue colour; the granules of the coarsely granular cell (*e*) have a greenish tint; the protoplasm of the cell containing basophile granules (*f*) is mauve.

Plate VIII.   Med. Chir. Trans., Vol. LXXXII.

A CASE

OF
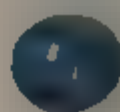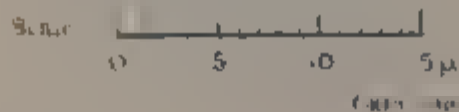
RHEUMATIC PERICARDITIS AND EXTRE] DILATATION OF THE HEART

WITH AN

INVESTIGATION INTO THE MICROSCOPY OF RHEUMATIC HEART DISEASE

BY

F. J. POYNTON, M.D.

COMMUNICATED BY DR. NORMAN MOORE

THIS case, for the use of which I am indebted to th kindness of Dr. Sidney Phillips, affords both clinical an pathological evidence in support of the statements mad by Dr. Lees and myself in two papers read before thi Society in June, 1898. The first, by Dr. Lees, empha sised the importance of acute dilatation of the heart i rheumatic fever; the second, a joint paper, dwelt upo the frequency of its occurrence in the rheumatism an chorea of childhood.

These views we supported by clinical observation

extending over a considerable period of time, and some of the results obtained were illustrated by cardiac tracings thrown on the screen by a lantern. Though personally assured of the truth of our clinical investigation, we felt it necessary to adduce pathological evidence in its support. This was to some extent supplied in an appendix to the papers, by an analysis of 150 cases of fatal rheumatic fever in children under twelve years of age.

In the discussion that followed, the great size of some of our cardiac tracings was criticised. Dr. Ewart, on the other hand, accepted in part the frequency of a considerable increase of the præcordial dulness, but considered the explanation to be an evanescent pericardial effusion.

This case is now brought forward because it deals to some extent with these difficulties, and demonstrates clearly the extreme dilatation that may occur in rheumatic pericarditis with no fluid in the pericardium and almost no valvulitis. The results of the microscopic investigations of the heart, which I have made in this case, seem to me to clearly indicate a morbid condition of its muscular structure, which goes far to explain the great dilatation.

The case is an example of aggravated cardiac rheumatism, minor degrees of which are of common occurrence. There will be, therefore, no need for lengthy clinical details, and those facts which are concerned with the condition of the heart will alone be described.

Arthur G——, aged 15, was admitted to St. Mary's Hospital on August 25th, 1898, for shortness of breath. In December, 1897, he passed through an attack of rheumatic fever in which several joints were swollen and very painful, but for which he did not keep his bed. In February, 1898, he had another attack of rheumatic fever, which lasted for two months. Since this last attack he had suffered from occasional pains over the heart on exertion. Two days previous to his admission to the hospital a small nodule appeared over the right patella.

On admission, on August 25th, his temperature wɛ 100° F., pulse 120, and respirations 30 ; he was very pa and emaciated. The cardiac area was increased, tl limits being *one* finger-breadth to the right of the rigl sternal margin, and *one and a half* to the left of tl vertical nipple line ; upward, the limit was the secon left intercostal space. The action of the heart wɛ excited, but no pericardial friction was detected. At tl apex there was a loud, harsh, systolic murmur conductɛ to the axilla, and at the base an accentuated pulmonar second sound. The next day pericardial friction wɛ heard, and this on the following days became loud an general. Upon August 30th the area of cardiac dulnɛɛ had increased, extending now *two* finger-breadths insteɑ of *one*, to the right of the right sternal margin, and sti *one and a half* beyond the left vertical nipple line. Th loud friction quite obscured the murmur. Upon Septem ber 2nd the friction was not so loud, but the cardiac dul ness reached the *right nipple line* and extended *two* fingeɪ breadths outside the left nipple line. Upon Septembe 3rd the friction was loud and general, and the cardiɑ dulness extended *one finger-breadth beyond* the rigɦ vertical nipple line, and about *three* beyond the lefɩ There was dulness in the fifth right intercostal spac (Dr. Rotch's sign). I was unable, even with the greatɛɛ care, to detect any curving inward of the right margin c the cardiac dulness at its lower limit. Over the front c the chest there was doubtful pleural friction on both sideɛ Upon September 7th the dulness extended *one inc* beyond the right vertical nipple line, and there wa systolic retraction of the right intercostal spaces. Perɩ cardial friction was still well marked. The liver extendeɩ two inches below the costal margin. Cough, vomitinɡ restlessness, and all the signs of acute cardiac failuɾ were apparent, and the patient died the following daɟ The temperature since the day of admission had nevɛ risen above 99·5°.

The *post-mortem* was made twenty-four hours aftɛ

death. During life the areas of cardiac dulness had been confirmed by Dr. A. G. Butler, Dr. Phillips's house physician. After death I verified the dulness as external to the right nipple, and far outside the left. This was also confirmed by a post-mortem assistant. Though the loud, general, and continuous pericardial friction negatived the idea of a large effusion, there seemed every probability that there would be a distinct increase in the amount of fluid, and I removed the chest wall with especial care, in order to obtain some of this fluid in Pasteur pipettes for bacteriological purposes. In this I was disappointed, for the pericardium was generally adherent, and the enlargement almost entirely due to the dilated heart, and, to a very minor extent, to the thickening of the pericardium. The area of dulness to the right of the sternum was due to the greatly dilated right auricle. In front, over a limited area, the pericardial adhesions were firm, but at the sides and behind the adhesions were quite recent, and the pericardium in places swollen to the thickness of half an inch. The recent adhesions were in the form of flakes of lymph, in the interstices of which was a little fluid. The two lungs were pushed aside by the immense heart, and were adherent to the pericardium by recent adhesions. The valvular changes were extremely slight, consisting only in some slight thickening of the mitral segments, not causing the least stenosis, and there was not any recent valvulitis. The valve rings were dilated, and the cavities of the heart crammed with pale and dark clot, and in the right auricular appendix the clot was found firmly adherent. Both ventricles seemed slightly hypertrophied. The weight of the heart with the pericardium, but empty of blood, was twenty-six ounces. The muscle was streaked with pale striæ, and had on section a pale purple colour. The other viscera showed changes compatible with a recent and acute cardiac failure.

I made numerous sections of the pericardium, heart-walls, blood-clot, and nodules, and will briefly describe

the salient points in the results obtained. (Some of th sections are shown under the microscopes.) In additioɪ I have taken sections from other examples of rheumati heart disease, from a case of advanced alcoholism Addison's disease, and from a case of cardiac fibrosis These appear to me to support the views that are held iɪ this paper, and they will be alluded to as the point they illustrate are considered.

In the case above described, a section through th inflamed pericardium showed extreme vascular dilatatioɪ and plastic inflammatory exudation into the pericardia cavity. In the wall of the left ventricle there was ʑ similar but slighter capillary distension, and both undeɪ the pericardium and far away from it there was a freɛ exudation of small cells between the muscle-fibres. Thɛ muscle itself showed great loss in striation, and many o the fibres showed granules in the proximity of the nucleus not of a fatty nature. In sections through the right ventricle, the most striking changes were beneath thɛ pericardium, but they were visible throughout the entirɛ thickness of the ventricle wall. Another series of sectionɪ were fixed in Hermann's osmic fluid. Both beneath thɛ pericardium and throughout the heart-wall there waɪ fatty degeneration of the muscle-fibres. Sections througl the right auricular appendix and *ante-mortem* clot showec externally pericarditis, then hyaline change in the muscle and coagulation necrosis in the blood-clot.

Thus it will be seen that the changes in the cardiaɛ wall were very general, affecting muscle, vessels, anɛ interstitial tissues. The early commencement of thɛ interstitial changes are, however, better seen in somɛ sections taken from a case of pericarditis of earlier date In this case, published in the 'Lancet,' July 23rd, 1898 death was caused by a rare complication, extensivɛ thrombosis of the large veins of the neck and uppeɪ extremities. The pericardial friction in this case waɪ detected only the day before death. The sections show numerous foci of inflammatory exudation scattered througl

the walls of the ventricle, and not localised to that part of the myocardium lying immediately beneath the pericardium. In this case also during life there was great dilatation.

Though these morbid changes are distinct and easily recognisable, the parenchymatous changes are by no means as profound in these rheumatic hearts as in some other conditions. Thus, in some sections taken from the left ventricle of a case of advanced alcoholism, it is difficult to recognise at first sight the muscular tissue, the changes are so extreme. Yet during life I especially noted that there was no appreciable dilatation, a point to which allusion will again be made. Again, though the alterations in the interstitial tissues of the heart are quite definite in these acute cases of rheumatism, they are more extreme in the sections of a fibroid heart taken from a case that terminated in the sudden death of the patient. Yet though the microscopic changes in these fatal cases of acute rheumatism were not extreme, they were general and diffuse.

The sections next described are also from a case of cardiac rheumatism, but with a different clinical history. They were taken from the left ventricle of a man aged twenty-nine. He had suffered in childhood from rheumatic fever, and five years before his death had been known to have organic, mitral, and aortic disease. He finally died in another attack of rheumatism, complicated with pneumonia. These sections show fibrosis of the ventricle—evidence, I believe, of the previous rheumatic attacks, and in addition multiple foci of small-celled exudation scattered through the ventricle—evidence of the recent illness. It is noteworthy that macroscopically this muscle appeared natural, and that there was not any recent valvulitis or pericarditis, but an adherent pericardium of old standing.

It remains now to consider shortly the bearing of these details upon the question of rheumatic dilatation. The case itself proves conclusively that great dilatation,

greater than any that was represented in our tracings last June, may occur in rheumatic fever with plastic pericarditis, but without valvulitis of any appreciable severity. It also proves that Dr. Rotch's sign may be obtained when there is no pericardial effusion. Such a case as this, taken together with the clinical evidence we brought forward in June, makes it probable that dilatation of less degree may occur without pericarditis, and that the myocardial changes are not a *sequela* of the pericarditis. If pericarditis is present the changes are concurrent, and they probably occur in the myocardium independently of any pericarditis.

It is, I believe, usually considered that the pericarditis is the starting-point of the myocardial inflammation, and is the explanation of the dilatation and severe symptoms that appear in the worst cases of cardiac rheumatism. Several observers have, however, held that the myo- and pericardial changes are concurrent, and part of the same rheumatic process. Thus, Dr. Cheadle, when writing of chronic pericarditis, suggests this in the Harveian Lectures on the "Rheumatic State in Childhood" in 1888, and the same opinion was expressed by the late Dr. Sturges in the term rheumatic carditis. Dr. Theodore Fisher, both at the meeting of this Society in June and again at Edinburgh in July, stated that he had observed pathological changes in the myocardium apart from any pericarditis.

The microscopic sections, I think, support the view that when pericarditis is present, the myocardium is also affected concurrently, for the changes in the heart-walls commence by numerous scattered foci, some of them far from the pericardium, and the changes in the muscle are general.

This view is also supported by the recent advances of pathology, especially in the investigation of cardiac muscle, for these impress the fact that micro-organisms gaining access to the body give rise to toxines which circulate in the system. These, doubtless, by delicate

chemical reactions injure the tissues, and usually have, to some extent, a specific action on certain classes of tissues. Thus the rheumatic poison appears to especially attack tissues which are in frequent movement or exposed to friction, for example, the heart, the joints, the tendons, the skin, and voluntary muscles. It is possible that these tissues have a certain chemical relationship which renders them thus liable. However this may be, when after death from rheumatic morbus cordis, acute changes are found throughout the heart, it is most probable that if rheumatism be caused by a toxine, these changes arose concurrently in a general infection of its structures.

Allowance, I think, must also be made for the influence of the surrounding tissue pressures. The pericardium, with a cavity between its two layers, and with numerous vessels and lymphatics lying in the deeper part of its visceral portion, will, in reacting to the rheumatic poison, permit free exudation into its sac, and after death the result is striking and at once appreciated. But in the ventricle wall the firm and contracting muscle will probably allow but little exudation, yet the morbid changes may in the gravity of their results be even greater. Post mortem we may find little macroscopically, and when we do find changes we are naturally inclined to consider that they are less frequent in their occurrence, and commence after the onset of the pericarditis. We have many proofs that the morbid results produced by a poison may be extreme, yet the obvious morbid changes in the tissues themselves may be slight, as, for example, in acute osteomyelitis or in tetanus.

Another point is one upon which Dr. Lees has frequently laid stress, and to which he recently called attention when introducing the discussion upon rheumatic heart disease in childhood at the Edinburgh meeting in July, 1898. It is that in rheumatism, dilatation is frequently very marked, yet the clinical symptoms, provided the patient be at rest, are often remarkably slight.

At first sight this is difficult to realise, but I believe

the explanation to lie in the fact that, the function of the cardiac muscle is damaged in a peculiar way rather than destroyed by the rheumatic poison.

A tissue so complex as the cardiac muscle is liable to a variety of pathological changes, and these probably have a different morbid significance. Some of these changes may be far more detrimental to the vital properties of the muscle than others. Those that are the most destructive may cause death from syncope without appreciable dilatation, as occurred in the case of alcoholism referred to above, and also in the case of Addison's disease. Those changes which are less destructive may cause perversion or impairment of function, ending perhaps in death, but with great dilatation of the heart during life.

Clinically, the first definite evidence of advanced fatty degeneration of the heart may be sudden death; and so we may expect that in rheumatism, with morbid changes of a less severe type, the symptoms due to the dilatation may be but slightly marked.

It is, I think, also legitimate to consider what would have happened had the case recorded in this paper made a temporary recovery. The pericardium, we know, would have become less hyperæmic, have shrunk, and finally have become generally adherent. It is more difficult to follow the fate of the muscle, but probably much would have been replaced by fibrous tissue. The sections through the ventricle of the case of recurrent rheumatism support this. If the boy had then died in a relapse, twelve months later, there would have been a temptation to lay much stress upon the mechanical effect of the obvious adherent pericardium, and little upon the loss of vital power due to the damaged muscle.

Finally, in rheumatic children it is certain that it is the active rheumatism that usually kills them. It is very probable that in adults also active rheumatism is of more importance than might be thought; and that the explanation of some cases of organic heart disease, which

fail to react to treatment, and in which the symptoms far
outweigh the clinical signs, is not the adherent peri-
cardium so often found after death, but this active
rheumatism injuring the cardiac muscle. The evidences
of this rheumatism may be slight, and possibly we do not
even yet recognise fully its manifestations. I have been
struck with the fact that not infrequently there is in
these unfavourable cases a history of recent rheumatism,
and now and again such definite symptoms as arthritis
develop during their stay in hospital. These symptoms
could then hardly be due to a fresh infection, and are
more probably an evidence of active rheumatism which
has existed from the time that the cardiac breakdown
brought them to the hospital.

Since this paper was communicated in February, I
have obtained microscopic sections of the cardiac muscle
in three cases, each of which show changes that bear
upon the questions considered in this paper. I am
indebted to Dr. Cheadle, Dr. Lees, and Dr. Phillips for
leave to give a brief outline of their histories.

Dr. Cheadle's case was that of a man of thirty, who
had suffered from rheumatic fever in childhood, and was
admitted for aortic and mitral incompetence. The sym-
ptoms were most urgent, quite overshadowing the physical
signs. The sections of the left ventricle showed much
fibrosis.

Dr. Lees' case, that of a girl of eighteen, was one of
acute carditis, almost precisely similar to the case recorded,
but even more convincing because there was no marked
thickening of the pericardium. The fatty changes
throughout the heart-wall were quite remarkable.

Dr. Phillips' case was that of a woman of twenty-
three, who died of "infective endocarditis," probably of
rheumatic origin. There were mural vegetations upon
the endocardium of the left ventricle. The ventricle-wall
showed *acute* inflammatory changes, but the pericardium
was completely adherent from a previous rheumatic attack.

I am indebted to Dr. Sanders for all the sketches that have been made of the sections that were shown.

(For report of the discussion on this paper, see 'Proceedings o the Royal Medical and Chirurgical Society,' Third Series, vol. xi p. 126.)

DESCRIPTION OF PLATE IX,

Illustrating a Case of Rheumatic Pericarditis and Extreme Dilatation of the Heart (F. J. POYNTON).

FIG. 1a. Diagram of pericardium and wall of the left ventricle (rheumatic pericarditis).

    a. Points to the pericardium.

    b. Points to the position in the ventricle from which the sketch shown in Fig. 1*b* was made.

FIG. 1b. Section of a portion of the left ventricle (rheumatic pericarditis).

    c. Points to distended capillaries.

    d. Points to cellular exudation between the muscle-fibres.

FIG. 2a. Diagram of pericardium and wall of the left ventricle (rheumatic pericarditis).

    a. Points to the inflamed pericardium.

    b. Points to the position in the ventricle from which the sketch shown in Fig. 2*b* was taken.

FIG. 2b. Section of a part of the left ventricle (rheumatic pericarditis).

    c. Points to a capillary cut longitudinally.

    d. Points to a free cellular exudation between the muscular fibres.

Plate IX.

Fig. 2a.

Fig. 1b

Fig. 1a.

Fig. 2b.

Fig. 2a.
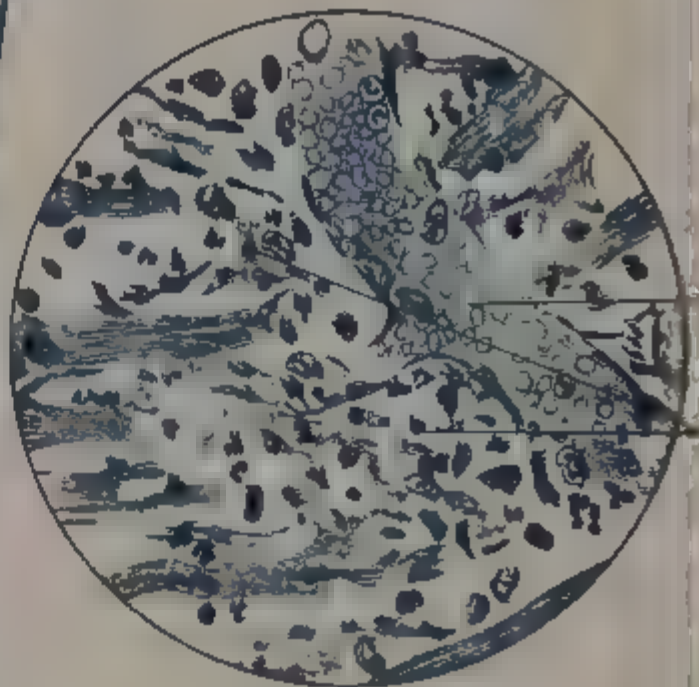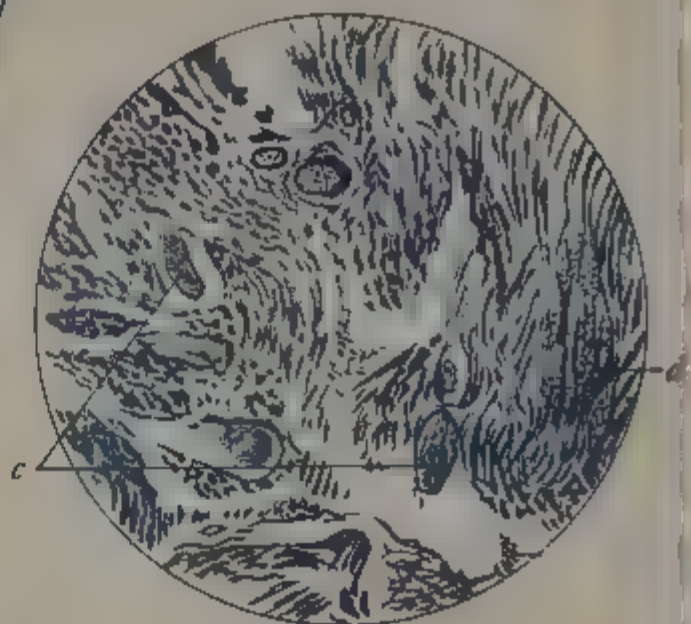
DESCRIPTION OF PLATE X,

Illustrating a Case of Rheumatic Pericarditis and Extreme Dilatation of the Heart (F. J. POYNTON).

FIG. 3. A section of a part of the left ventricle just beneath the pericardium (rheumatic pericarditis).

> a. Points to the dark dots which represent fatty changes in the muscle-fibres. Hermann preparation.

FIG. 4. A part of the same ventricle wall as that represented in Fig. 3, but close to the endocardium.

> a. Points to the fatty changes in the muscle-fibres. Hermann preparation.

FIG. 5a. Section through pericardium and wall of the left ventricle in a case of early rheumatic pericarditis, fatal by severe venous thrombosis.

> a. Points to the pericardium.
>
> b, c, d. Point to foci of cellular exudation spreading from the blood-vessels.

FIG. 5b. One of the foci seen in Fig. 5a, more highly magnified.

> a. Points to cellular exudation between the muscular fibres.

Plate X.   Med. Chir. Trans., Vol. LXXXII.
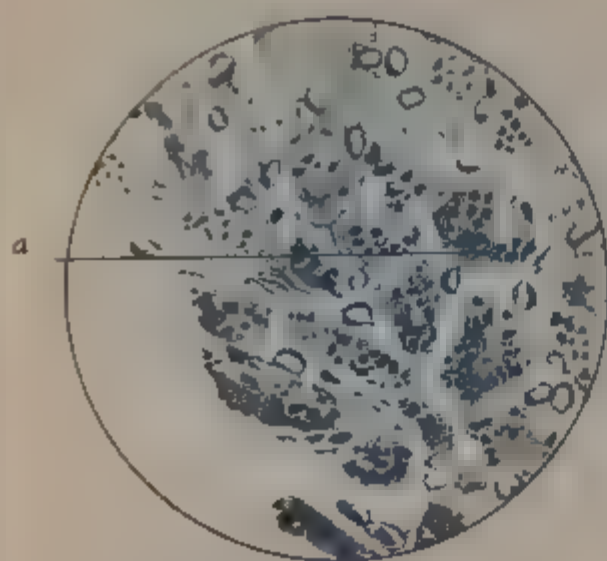
Fig. 3.
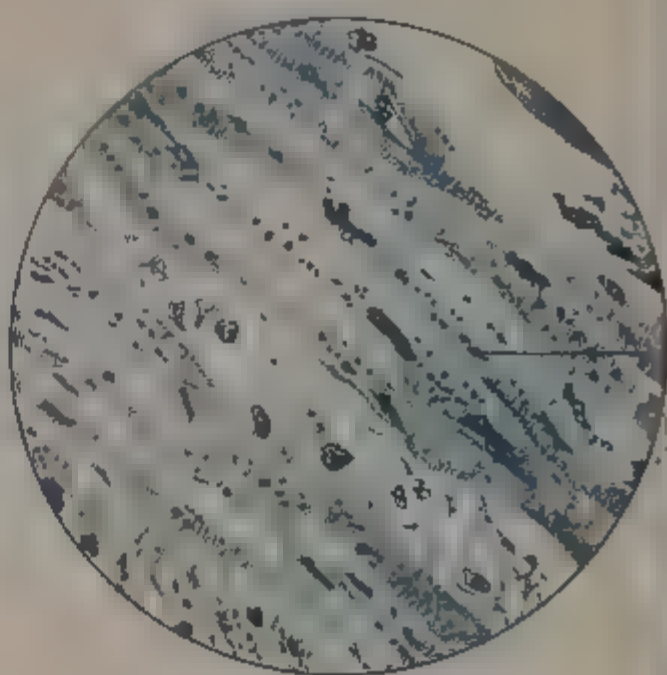
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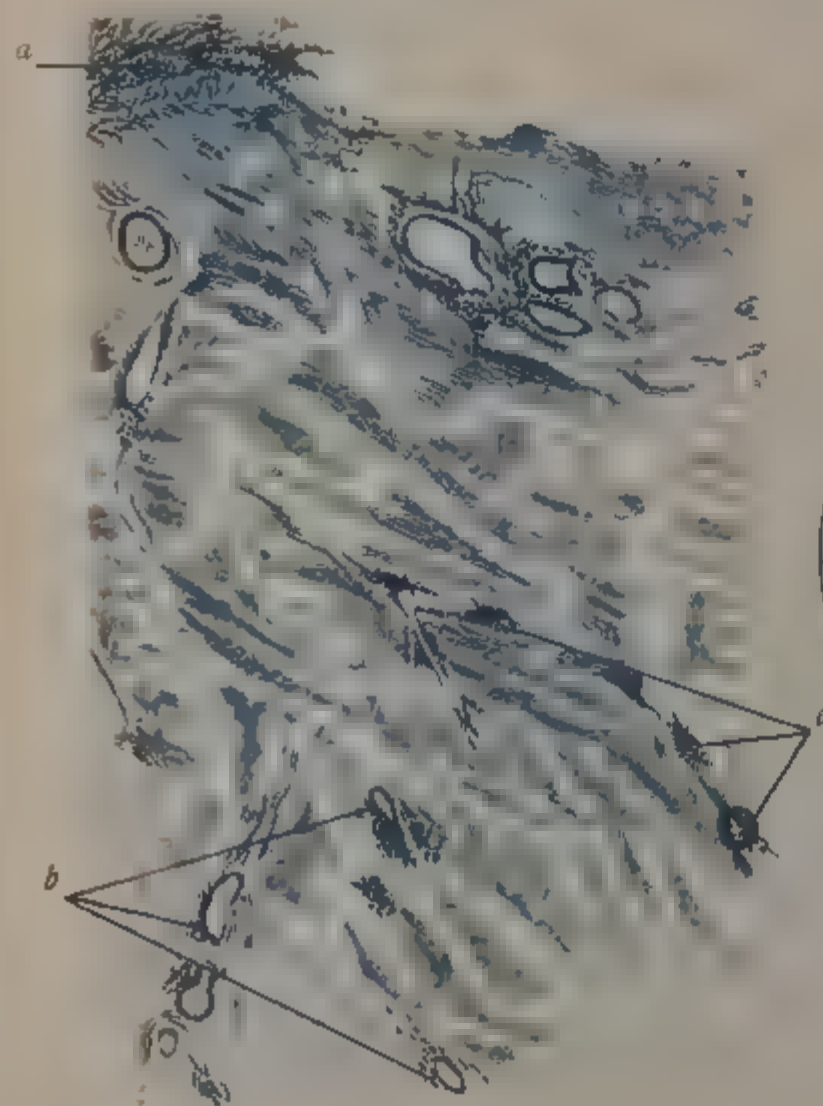
Fig 5a.
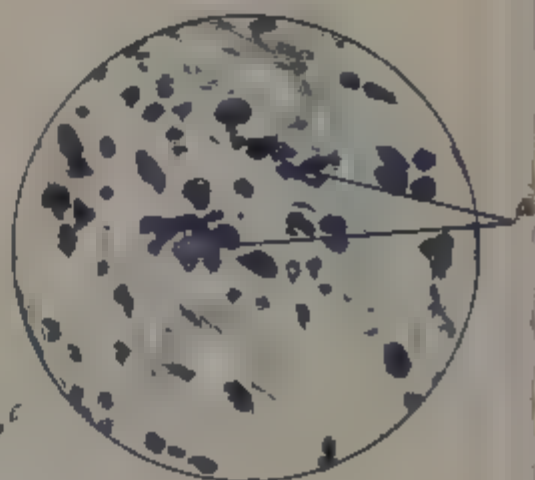
Fig 5b.

DESCRIPTION OF PLATE XI,

Illustrating a Case of Rheumatic Pericarditis and Extreme Dilatation of the Heart (F. J. POYNTON).

FIG. 6. Section of a part of the left ventricle from a case of recurrent rheumatism.

     a. Points to cellular exudation between the muscular fibres.

     b. Points to a fibrous strand, spreading from the vessels and implicating adjacent muscular fibres.

FIG. 7. Section of a part of the same left ventricle as Fig. 6, under higher magnification.

     a. Points to cellular exudation.

     b. Points to hyaline change in muscle-fibres.

FIG. 8. Section of a part of the left ventricle from a case of chronic alcoholism.

     a. Points to a muscle-fibre still recognisable.

     b. Points to a nucleus of a muscle-fibre.

     c. Points to fatty changes in a muscle-fibre.

     d. Points to spaces left by the complete destruction of the muscle-fibres. Hermann preparation.

Plate XI.   Med. Chir. Trans., Vol. LXXXII.
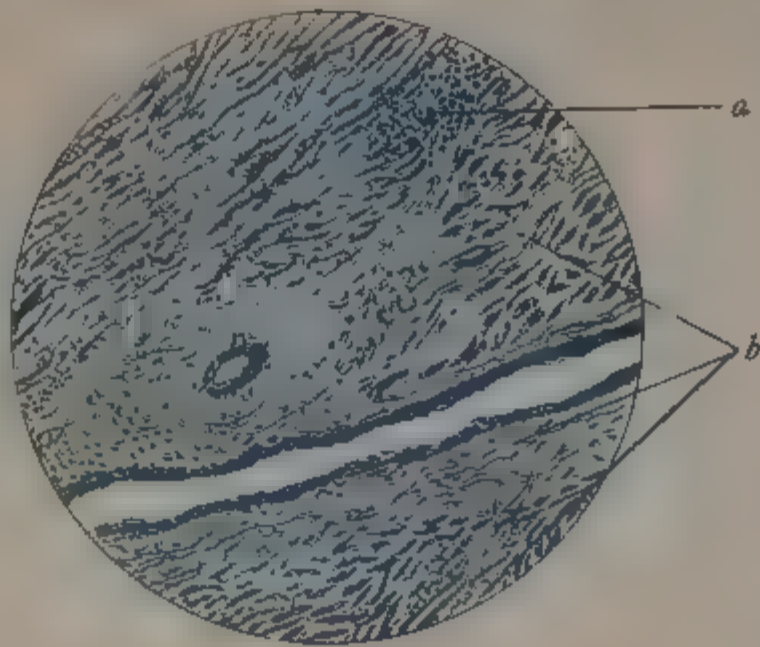
Fig. 6.
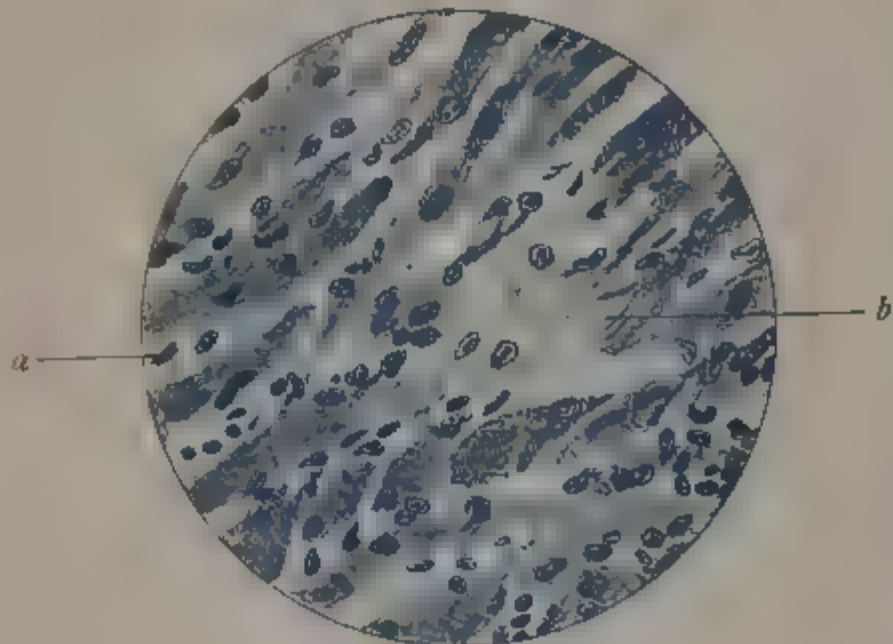
Fig. 7.
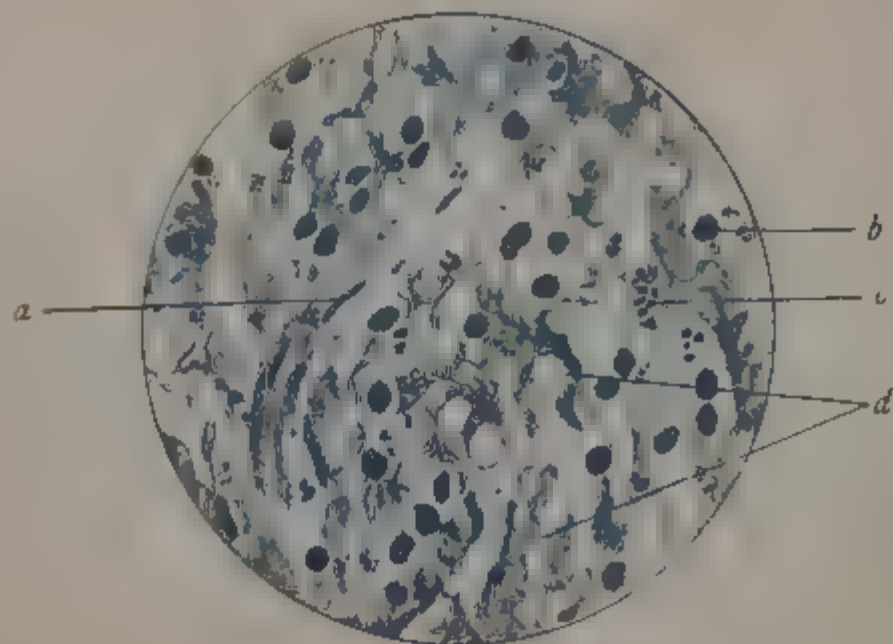
Fig. 8.

DESCRIPTION OF PLATE XII,

Illustrating a Case of Rheumatic Pericarditis and Extreme Dilatation of the Heart (F. J. POYNTON).

FIG. 9. Section from the left ventricle of a man aged 30, who had suffered from rheumatic fever in childhood.

    a. Points to the muscle-fibres.

    b. Points to coarse strands of fibrous tissue traversing the ventricle.

FIG. 10. Section of a papillary muscle from the same case as Fig. 9.

    a. Points to fibrous strands between the muscle-fibres.

    b. Points to the muscle-fibres.

    c. Points to fibrosis around a blood-vessel.

Plate XII.   Med. Chir. Trans., Vol. LXXXII.
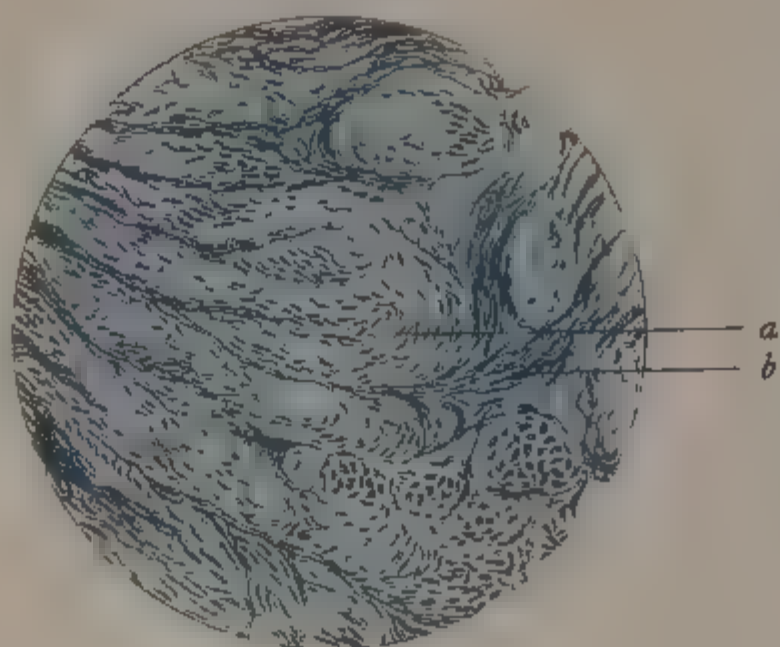
Fig 9.
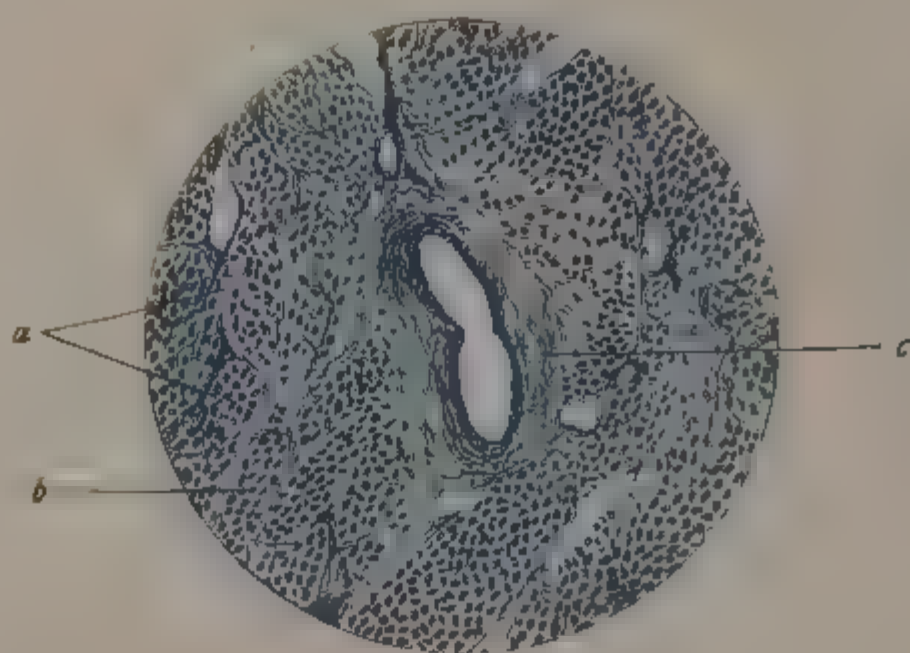
Fig. 10.

A CONTRIBUTION

STUDY OF ALKAPTONURIA

BY

ARCHIBALD E. GARROD, M.A., M.D.

THE rare anomaly which is known as alkaptonuria possesses great interest alike for the physiologist and the physician; for the former because the study of the abnormal constituent or constituents which are present in the urine of alkaptonuric individuals, and of the conditions which control their excretion, promises to throw important light upon the metabolism of proteids and upon the fate of tyrosin in the organism; for the latter because the condition is one liable to be mistaken for glycosuria, and because of the peculiar coloration of the urine which develops on standing, and on account of which a considerable proportion of the cases come under observation.

The following are the essential features of the urine of alkaptonuria:

1. Although of normal appearance when passed, the urine rapidly acquires a deep brown colour and ultimately becomes black on exposure to air.

2. The brown colour is greatly intensified by alkalies, its development being accompanied by absorption of oxygen.

3. The urine reduces Fehling's solution with the aid of heat, and actively reduces ammoniacal silver nitrate solution in the cold.

4. The bismuth test for sugar yields negative results (one recorded example alone offered an exception to this rule).

6. The urine has neither dextro-rotatory nor lævo-rotatory power.

7. The fermentation test yields negative results.

8. Fabrics moistened with the urine become deeply stained on exposure to air.

The Clinical Aspects of Alkaptonuria.

In the table on page 392 I have entered all the recorded cases of alkaptonuria which I have been able to find, and in which the necessary details are forthcoming, commencing with the case recorded by Bödeker[1] in 1859 in a paper in which he described the earliest attempt to isolate the substance to which the reducing power of such urines is due.

In the fourth edition of Bowman's 'Medical Chemistry,' edited by Bloxam (1862), it is mentioned that Dr. Johnson had observed the occurrence of alkaptonuria in an infant, his attention being called to it by the staining of the napkins. No further details are given, and I have been unable to find any fuller record of the case, which would rank second in chronological order. It is therefore not included in the table.

Nor has a search in much earlier literature proved wholly fruitless. In 1822 Dr. Alexander Marcet read before the Medico-Chirurgical Society an account of the urine of an infant, aged seventeen months, which turned

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[1] References to the authors quoted will be found in the bibliography at the end of the paper.

black soon after being passed, and which stained napkins of a dark purple hue. The condition was noticed almost immediately after the child's birth. The darkening effect of alkalies was specially noted. The chemical properties of the pigment, which were investigated by Prout, agreed with those of the alkapton pigment, and, although evidence of a reducing action of the urine is wanting, it seems hardly possible to doubt that this was a case of alkaptonuria.

Schenck, in his 'Observationes Medicæ,' published in the seventeenth century, in a chapter dealing with the passage of black urine by healthy persons, quotes from Johan Bellfortis, the case of a monk who passed black urine during the six years that he was under observation, and stated that he had done so all his life. This also may have been an example of the condition under consideration.

In addition to the twenty-four recorded cases, there are included in the table seven cases hitherto unpublished. For the notes of five of these I am greatly indebted to Dr. Pavy, who also kindly supplied me with some specimens of urine.

Of the remaining patients one is an infant who came under the care of my colleague Dr. Voelcker, at the Hospital for Sick Children, and to him I would here express my gratitude for his kindness in handing over the case to me.

The last patient is under the care of Dr. C. E. Baker, to whom also my best thanks are due for the particulars given, and for furnishing me with several litres of the urine for examination.

It is upon an analysis of these thirty-one cases that the following account of the clinical features of alkaptonuria is based.

Alkaptonuria would appear to be considerably commoner in males than in females, the series of thirty-one

cases including twenty-three males and only eight females.

In the great majority of instances the anomaly dates from early childhood, but it has occasionally appeared as a temporary phenomenon during an illness, or has apparently developed in later life. In a certain number of the recorded cases no data bearing upon this point are forthcoming, or it is merely stated that the alkaptonuria was of long standing. In seventeen cases it had been observed from infancy, but it is probable that the above figure does not at all represent the full proportion of life-long cases. In five cases only is the phenomenon definitely stated to have been merely temporary or of later development.

In the case of my own patient the staining of the napkins was noticed on the day following birth, and in Ebstein and Müller's case (No. 3) the alkaptonuria was traced back to the early days of life.

In one instance, Stange's (No. 18), the phenomenon was apparently intermittent, the urine losing its abnormal properties for long periods, and this observation renders it most important that such intermittent alkaptonuria should be, as far as possible, excluded in cases apparently of later development. Evidence upon this point is not always easy to obtain, and in at least one case, Ogden's (No. 17), the patient, an adult man, was unaware of any peculiarity of his urine.

In this connection, evidence supplied by the mothers of patients as to staining of the napkins in infancy is of special value.

Life-long alkaptonuria is sometimes met with in several members of a family, but, although brothers and sisters are apt to share this peculiarity, I know of no instance in which it has been transmitted from one generation to another. When it occurs in families some members are apt to escape, and a child born between two alkaptonuric members may pass normal urine.

Kirk's patients (Nos. 7, 8, and 9) were three out of

four brothers of whom the eldest escaped. Marshall and Futcher's patients (Nos. 10 and 22) were brothers. Walter Smith's patient (No. 5) has an alkaptonuric brother (No. 26) whose urine I have had the opportunity of examining. An intermediate brother has escaped. Baumann and Embden's patients. (Nos. 12 and 13) were a brother and sister born out of wedlock. Both parents subsequently married and both had children, none of whom were alkaptonuric.

Dr. Pavy has met with a family of fourteen, amongst whom were four alkaptonuric members (Nos. 28, 29, 30, and 31). These were the ninth, eleventh, thirteenth and fourteenth children. The tenth and twelfth had escaped.

As Wolkow and Baumann pointed out, life-long alkaptonuria is apparently without any injurious effect upon the health of its subjects. Baumann's patient had reached the age of sixty-eight, and his sister, whose urine was studied by Embden, that of sixty years.

The staining action of the urine is usually the only source of annoyance, unless the patient presents himself for life insurance and, as has repeatedly happened, is rejected as diabetic.

Stange's patient, who excreted an exceptionally large amount of the abnormal constituent, suffered from dysuria and herpes of the penis, but my patient, although his urine is concentrated, scanty, and very rich in the same substance, has no dysuria, although frequent scanty micturition is present. In one instance (No. 5) slight ailments were apparently attended by more intense darkening of the urine, and in one of Dr. Pavy's cases the urine was noticed to be darker than usual before the menstrual periods.

The temporary cases are of great interest, and, curiously enough, of the five such cases recorded no less than four have been in females.

Carl Hirsch's case (No. 21) is specially striking because it was definitely stated that the urine had never previously shown the same characters, and there was no other known

case in the family. The patient, a girl aged 17, who had suffered from no previous illness except measles, passed urine having the characteristic features of alkaptonuria on three successive days, during an attack of gastro-enteric catarrh with diarrhœa and vomiting. The phenomenon did not recur during convalescence. A chemical examination of the urine was made by Professor Siegfried, and, although conclusive analytic data are not furnished, there is practically no doubt as to the nature of the abnormal substance present.

W. von Moraczewski's patient (No. 19) was a woman aged 43, whose urine was of natural appearance on her admission to hospital with phthisis and tubercular peritonitis. Alkaptonuria developed shortly before death, and no history of previous alkaptonuria could be obtained.[1]

Geyger's case (No. 14), of which hardly any clinical details are given, was very remarkable. A diabetic man passed alkapton urine on one day only, and the diagnosis was fully confirmed by chemical examination. The abnormal constituent was afterwards sought for in vain for weeks, but we learn from a statement of Embden's that it was again found on a subsequent occasion after the sugar had disappeared from the urine.

In Slosse's case (No. 16) the development of alkaptonuria apparently coincided with an aggravation of the patient's malady. The patient was a woman, whose age is not stated, who suffered from pyonephrosis for which an operation was performed. The case ended fatally.

Maguire's case (No. 6) is in some respects unique. The patient, a female, who had suffered from gastric ulcer two years previously, developed alkaptonuria at the age of 27, six months before Maguire's examination of the

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[1] Galloway recently mentioned, in a discussion at the Dermatological Society of London, the case of a woman, aged about thirty-five, who developed what was in all probability alkaptonuria as a præmortal symptom in the course of general desquamative dermatitis. The urine reduced Fehling's solution, yielded no osazone, and developed a brownish tint on exposure to air.

urine was made. Apparently its development did not coincide with any illness, and when developed it con-tinued at any rate for months.

The Chemistry of Alkaptonuria.

Bödeker, who in the year 1859 first described alkap-tonuria, isolated the peculiar reducing substance in a crude condition, and, on account of its behaviour with alkalies, assigned to it the name of alkapton (alkali κάπτειν). He believed it to be a nitrogenous compound In Bödeker's case, as in one subsequently recorded by Geyger, sugar was also present. Although, as the result of the later researches of Kirk, Wolkow and Baumann, Huppert and others, we now possess a far more accurate knowledge of the chemistry of the condition, the name "alkaptonuria" persists, and is likely to persist, as a convenient designation for the anomaly which Bödeker described.

In 1875, Ebstein and Müller, whose patient was an infant, concluded from the reactions of the urine that the reducing substance present was pyrocatechin, and Fleischer arrived at a similar conclusion from his investi-gation of an adult case.

In 1882, Walter Smith, who examined the urine of an alkaptonuric child, concluded that the abnormal con-stituent was not pyrocatechin but more probably proto-catechuic acid.

In 1886, Kirk, who had the opportunity of studying two cases in a family of which a third member had also been alkaptonuric, isolated a substance which he then called urrhodic acid, but which he subsequently showed (1888) to contain two distinct substances, to which he assigned the names of uroleucic and uroxanthic acids respectively. Ultimate analyses of uroleucic acid led him to assign to it the formula $C_9H_{10}O_5$.

In 1887, Marshall isolated from an alkaptonuric urine

by a different process, a crystalline acid which he called glycosuric acid.

In 1891, Wolkow and Baumann extracted from a similar urine a crystalline acid to which the reducing action of the urine in question was apparently wholly due, and to which they assigned the name of homogentisinic acid. Analyses showed that it had the formula $C_8H_8O_4$, and it was apparent, from an elaborate study of its reactions and decompositions, that it must be regarded as hydroquinone-acetic acid. They devised a method for its quantitative estimation in which advantage was taken of its power of reducing ammoniacal silver nitrate in the cold. Baumann and Fränkel afterwards synthesised homogentisinic acid from gentisinic aldehyde.

We learn from Embden that Baumann obtained homogentisinic acid from some of Marshall's material, and Huppert, whilst confirming Kirk's observations as regards the presence of uroleucic in his cases, found that in some crude material sent to him by Kirk homogentisinic acid was present in still larger amount than uroleucic.

From his investigation of uroleucic acid Huppert arrived at the conclusion that of two possible compounds corresponding to the empirical formula arrived at by ultimate analysis of this substance, viz. trioxybenzene-propionic acid and dioxybenzene-lactic acid, uroleucic acid is in all probability the latter.

Geyger, who was unacquainted with Baumann and Wolkow's results, then recently published, obtained an acid which he regarded as identical with the glycosuric acid of Marshall, but the results of analyses of the lead salt which he gives agree very well with those of homogentisinate of lead. All other subsequent observers who have investigated cases of alkaptonuria have demonstrated the presence of homogentisinic acid, and have supported their identification of this substance by evidence of varying degrees of cogency.

Thus we find two distinct aromatic acids, homogentisinic and uroleucic acids, occurring in the urine of

alkaptonuric subjects. Of these, homogentisinic acid has been found in every case in which it has been looked for, and has been shown to have been present in some of the cases described before the classical research of Baumann and Wolkow. Uroleucic acid, on the other hand, has hitherto been met with only in Kirk's cases, but, except in Baumann's case, its presence does not appear to have been excluded. It is not improbable that this acid, which resembles homogentisinic acid in its properties, and which is much less readily detected, may have been present in other urines besides those examined by Kirk, and it is desirable that all future cases should be investigated with a view to demonstrating or excluding its presence.

The Nature of Alkaptonuria.

Our knowledge of the nature of the anomalous process which leads to the excretion of the alkapton acids in these rare instances is still scanty, but Wolkow and Baumann demonstrated the remarkable fact that the administration of tyrosin, by the mouth, to an alkaptonuric individual greatly increases the excretion of homogentisinic acid in the urine, although tyrosin given by the mouth does not cause alkaptonuria in a healthy individual. Indeed, by far the greater part of the tyrosin given to their patient reappeared in the urine in the form of homogentisinic acid. Embden repeated this observation upon a second patient, a sister of the preceding one, and obtained a similar, although not quite so conspicuous, increase.

In both patients, as in Ogden's and Stange's cases also, a meat diet conspicuously increased the excretion of homogentisinic acid.

Wolkow and Baumann produced alkaptonuria in a dog by the administration of 4·5 grammes of pure homogen-tisinic acid by the mouth, but a considerable portion of the acid administered was apparently broken up into

tolu-hydroquinone and carbonic acid, and there was a conspicuous increase of the aromatic sulphates in the urine. Embden's experiments upon himself showed that 4 grammes of homogentisinic acid taken by the mouth did not appear at all in the urine, whereas 8 grammes caused a temporary alkaptonuria and intolerable dysuria, but only a small proportion of the amount taken appeared in the urine. In the case of his alkaptonuric patient, on the other hand, 75 per cent. of a dose of homogentisinic acid (10 grammes) given by the mouth reappeared in the urine.

Unlike tyrosin, phenyl-acetic and phenyl-amido-acetic acids when administered to Embden's patient caused no increased excretion of homogentisinic acid. Ogden obtained a similar negative result with benzoic acid.

The above observations seem to show conclusively that tyrosin is the parent substance of the homogentisinic acid in the urine, but, as Wolkow and Baumann point out, beyond the fact that they are both aromatic acids these two substances have nothing in common, and the conversion of the former into the latter involves a shifting of the hydroxyl groups upon the benzene ring, in other words a simultaneous oxidation and reduction within the molecule. Such a change is, as they further point out, unknown as a metabolic process within the tissues, whereas it is observed as the result of fermentative changes. Hence, by a line of argument for which I must refer my readers to the original paper, they arrive at the conclusion that it is probable that alkaptonuria is due to the bacterial activity in the intestines, and perhaps to the presence of a special bacterion in such cases. The difficulty presented by the rarity of the condition they meet by a comparison with that known as *Diaminuria*, a condition almost equally rare and which also may persist for long periods. Embden and several more recent observers have initiated experimental investigations with a view to testing the correctness of this theory, but hitherto the results obtained, if

not directly opposed to Baumann and Wolkow's view, have failed to lend it any support.

Neither Baumann and Wolkow nor several more recent observers have succeeded in detecting any trace of the alkapton acids in the fæces, even in the diarrhœal stools after castor oil, which may be supposed to represent the contents of the upper portions of the intestine.

Embden, Ogden and others have tested the effects of various intestinal antiseptics, including β-naphthol, oil of turpentine and kefyr, but although conspicuous diminution of the ethereal sulphates of the urine was observed, the excretion of homogentisinic acid remained unaltered.

Culture experiments from the ordinary stools and from those passed after the administration of castor oil have yielded negative results, no alkapton acid being formed either by cultures in broth, meat juice or tyrosin broth.

It must therefore be acknowledged that the weight of evidence is at present opposed to the views of Wolkow and Baumann; and Embden's observation, that when an alkaptonuric individual took homogentisinic acid by the mouth a far larger proportion reappeared in the urine than when the same substance was administered to a healthy individual, would seem to point to abnormal metabolism in the tissues, in spite of the difficulty of reconciling the chemical processes here involved with the known facts of tissue-metabolism.

Stier obtained from the abundant aural wax of his patient a substance which had the properties of an alkapton acid, but no other observations which are recorded point to the occurrence of homogentisinic acid elsewhere than in the urine. That it is absent from the fæces has already been mentioned, and Fürbringer failed to obtain the characteristic reactions in the pericardial effusion, decolourised blood, or in a watery extract of the kidneys in one of the earliest observed cases.

I must here mention that it has been repeatedly observed that alkapton urines do not deposit crystals of uric acid even after the addition of an acid, and some

observations by Embden, Ogden, and Stange, seemed to show that in these cases the excretion of uric acid is conspicuously diminished. As all silver methods are inapplicable to these urines, the observers mentioned employed Fokker's method for the estimation of uric acid.

More recent estimations by Futcher, Stier, and Noccioli and Domenici, who all employed the more accurate method introduced by Hopkins, showed no such diminution, and an observation of my own points in the same direction, as will be presently shown.

Stier's observations are specially conclusive, as he made daily comparative estimations of the uric acid excretion of an alkaptonuric boy, aged 8, and that of his brother, aged 9, whose urine was normal. Sometimes the one and sometimes the other passed the larger amount of uric acid.

Moreover, alkapton urines *may* deposit crystals of uric acid which are deeply stained by the brown pigment. Ebstein and Müller noticed this and Stange observed crystals on some days and not on others ; again, one of the urines which I have examined deposited dark brown crystals of uric acid on standing.

After this preliminary summary of the present state of our knowledge of alkaptonuria I will proceed to the description of my own observations.

CASE 1. Thomas P— came under Dr. Voelcker's care, at the Hospital for Sick Children, at the age of three months. He was brought to the hospital on account of the peculiar appearance of his urine, which acquired a deep reddish-brown colour and stained the napkins deeply. The stains became developed on exposure to air. The mother stated that the woman who nursed her in her confinement called her attention to the staining of the napkins on the day following the birth of the child.

At that time the urine and the stains had a much redder or more purple colour than they have at present (the child being now more than a year old), and a similar

change of tint after the earliest months of life was observed by Ebstein and Müller in their case.

The child was fed entirely by the breast to the age of ten months, was fairly well nourished and exhibited no impairment of his general health. He was stated to have suffered from inflammation of the lungs when two months of age. He is the youngest of four children ; the mother is fairly strong, but the father, a drayman, is delicate and complains of his chest. No other case of similar peculiarity of the urine has occurred in the family, and I have examined the urine of the three elder children with negative results.

After weaning, at the age of ten months, the child was admitted as an in-patient, and I am indebted to Dr. Barlow for kindly allowing him the use of a cot in his ward.

After a stay of three weeks in hospital the child was discharged, but was shortly readmitted with a severe attack of summer diarrhœa which at one time threatened to prove fatal, but from which he made a good recovery, and has since enjoyed excellent health, save for troubles incidental to teething.

Careful inquiry from the mother failed to elicit any account of staining of the body clothing of the patient, such as might be expected to occur if homogentisinic acid had been present in the sweat, and no such staining was observed in the hospital, although the urine stained deeply any fabric wetted with it.

The fæces were repeatedly examined, but neither alcoholic nor watery extracts ever gave any alkapton reactions. Dr. Drysdale, who was good enough to make a bacteriological examination of a specimen of fæces obtained just before the child left the hospital, reports as follows :—
" Gelatine and agar cultures were made from the fæces, but very few organisms grew. In the second and third dilutions only one organism grew. This was a diplococcus which grew slowly on gelatine, forming round, raised colonies of a Chinese-white colour. It grew

equally well under aërobic and anaërobic conditions. It did not form gas nor coagulate milk. When grown in ordinary broth to which tyrosin had been added no substance yielding the alkapton reactions was formed either in aërobic or anaërobic cultures. No animal experiments were performed. This diplococcus is not, in my experience, a familiar organism of fæces; its predominance in the cultures was remarkable, but, as only one specimen was examined, its presence may have been accidental."

Cultures made on a subsequent occasion showed only the ordinary intestinal organisms with an abundant growth of *Bacillus coli communis*.

The urine of the patient is unusually scanty in amount, always acid in reaction, and highly concentrated. On some occasions the specific gravity has been as high as 1035. When the urine is freshly passed its colour is natural, but it quickly darkens, assuming a deep brown tint. It reduces Fehling's solution with the aid of heat, but the reduction is capricious and is considerably masked by the blackening due to the alkaline reagent. Alkalies greatly intensify the brown colour. Ammoniacal silver solution is readily reduced in the cold. The urine does not rotate the polarised ray in either direction, and no reduction of specific gravity or evolution of gas is produced by yeast. The bismuth test for sugar gives a negative result.

Micturition, though scanty and unusually frequent, is apparently not painful.

The youth of the patient precludes for the present any systematic analyses of the daily excretion, but I hope to make such observations at a later period.

No uric acid crystals are deposited from the urine on standing, even after the addition of an acid, but the amount of uric acid excreted is certainly not unduly small. On one occasion, when almost the entire quantity of urine passed during twenty-four hours was collected, 100 c.c. were found by Hopkins's method to contain 0·08075 grm. of uric acid. As the total amount of urine

was only 120 c.c., this corresponds to a minimum day's excretion of 0·0969 grm. of uric acid.

From a healthy child, aged one year, 400 c.c. of urine were collected in twenty-four hours, and the total amount of uric acid contained therein was found to be 0·063 grm.

For the detection of homogentisinic acid in the urine two different methods were employed. Firstly that of Wolkow and Baumann, as modified by Ogden, which may be briefly summed up as follows:[1] Acidification of the urine with dilute sulphuric acid; repeated extraction with equal volumes of ether; distillation of the ether; solution of the syrupy residue in water, and addition to the solution when heated nearly to boiling of a concentrated solution of basic lead acetate. On cooling, acicular or prismatic crystals of lead homogentisinate are deposited and continue to form for at least twenty-four hours.

On applying this method to the urine in question a rich formation of crystals took place, which had the characteristic form, and were, as is always the case, tinted by included pigment. When the crystals were dissolved in hot water the solution gave the alkapton reactions, and yielded, as solutions of homogentisinic acid should do, a transitory dark blue colour with a dilute solution of ferric chloride.

0·1923 grm. of the crystalline product lost, at 100° C., 0·0175 grm. of water of crystallisation = 9·10 per cent. The calculated loss for the formula $(C_8H_7O_4)^2Pb.3H_2O$ = 9·08 per cent.

Again, 0·2268 grm. of the dehydrated crystals yielded, after repeated treatment with sulphuric acid in a platinum crucible to constant weight, 0·1263 grm. of lead sulphate, corresponding to 38·03 per cent. of lead; whereas the calculated percentage of lead in dried lead homogentisinate is 38·25.

A specimen of the lead salt was decomposed, under water, by means of a stream of sulphuretted hydrogen,

[1] For details of the process I must refer my readers to the original papers.

and, after the excess of sulphuretted hydrogen had been boiled off, the filtrate was evaporated to dryness at a low temperature. Crystals of homogentisinic acid were obtained which were purified by re-crystallisation from ether. The colourless silky prisms of the free acid were dried in the exsiccator, which caused them to become white and opaque, and were found to melt between 145° and 146° C. (The melting point of homogentisinic acid is 145° to 147°.)[1]

The free acid is somewhat hygroscopic and, in order to obtain satisfactory determinations, it requires to be thoroughly dried, as otherwise the presence of water materially lowers the melting point.

The above observations showed beyond all question that homogentisinic acid was abundantly present in the urine of Thomas P—.

Considerably larger yields of lead homogentisinate were obtained by an extremely simple method of extraction, which I have described in a recent number of the 'Journal of Physiology.' I found that when the urine itself was heated nearly to boiling, without any preliminary treatment, and for each 100 c.c. at least five or six grammes of solid neutral lead acetate were added, after filtering off the bulky precipitate which was formed, the clear yellow filtrate deposited crystals of lead homogentisinate on standing.

The formation of crystals under these circumstances usually commences within two or three hours, and is complete in twenty-four hours. Cold greatly hastens the separation, but does not materially increase the yield.

By this simple process the urine of Thomas P— yielded fairly constantly about 0·5 grm. of the lead salt for each 100 c.c. In applying the process to urines less rich in homogentisinic acid larger quantities of lead acetate may require to be added, and in summer weather artificial

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[1] I am indebted to Dr. Orton, Demonstrator of Chemistry at St. Bartholomew's Hospital, for the loan of apparatus, and for kindly checking the various determinations of melting points.

cooling will perhaps be necessary to start crystallisation.

A comparison with the urine of a second patient showed that homogentisinic acid was present in this urine in unusually large amount, although owing to the scanty excretion the daily total was not remarkable. A specimen in which the reducing power was estimated by Wolkow and Baumann's silver method was found to reduce a quantity of silver nitrate equivalent to 0·0577 grm. of homogentisinic acid per 10 c.c., and this also is an unusually high figure. However the reducing power of uric acid introduces, as Morner has shown, a considerable error, and, as has been mentioned, this urine was by no means deficient in uric acid.

It remained to be ascertained whether or no the uroleucic acid of Kirk was also present in this urine. In searching for it I followed a plan similar to that recommended by Professor Huppert in the tenth edition of Neubauer and Vogel's work, and would take this opportunity of thanking Professor Huppert, not only for his kind replies to questions, but also for some valuable suggestions derived from his most recent observations which are not yet published.

Professor Huppert's plan is based upon the fact that uroleucic acid is not thrown down as a lead salt by the addition of such quantities of lead acetate as are employed in the processes described above, although Kirk succeeded in obtaining the lead salt by rubbing up an excess of neutral lead acetate with the solution until a thin paste was formed.

Any uroleucic acid which may be present is therefore to be looked for in the mother liquor from which the crystalline lead homogentisinate has been deposited. Unfortunately the precipitation of homogentisinate is not *complete* when either Wolkow and Baumann's or my simple method is employed, and consequently a complete separation of the two acids cannot be brought about by such means.

Uroleucic, like homogentisinic, acid gives the various alkapton reactions, but with ferric chloride a green instead of a blue colour appears, and uroleucic acid melts at 130° to 133° C., whereas the melting point of homogentisinic acid is 145° to 147° C. If, then, the remainder of the alkapton acid can be extracted from the mother liquor, the melting point will supply a means of detecting uroleucic acid, seeing that the melting point of a mixture of the two acids should be lower than that of either of the constituents of the mixture.

The chief difficulty met with is in the adequate purification of the small specimens of residual acid obtained, for the crystals obtained by evaporation of the ethereal extracts are always embedded in a brown syrupy material which is taken up by the same solvents as the alkapton acids.

In my earlier attempts extracts obtained by Baumann and Wolkow's method were employed. I found that even when all crystallisation had ceased and further addition of basic lead acetate caused no more crystals of lead homogentisinate to form, the mother liquor, which should contain any uroleucic acid present, still contained traces of alkapton acid, but it still gave with ferric chloride a dark blue colour, whereas uroleucic acid is described as yielding a transitory green tint with that reagent. After removal of the lead by means of a stream of sulphuretted hydrogen, and evaporation of the filtrate at a low temperature, no adequate amount of crystalline acid was obtained for a satisfactory purification and determination of the melting point.

The filtrate obtained in the simple process, after lead homogentisinate had ceased to crystallise out, was next treated as follows :—Sulphuric acid was added in sufficient quantity to precipitate the excess of lead in the form of sulphate and to leave the filtrate acid ; the acidified liquid was then repeatedly shaken with equal volumes of ether ; the ether was distilled off, and the brown syrupy residue was placed in a small dialyser immersed in water

with a view to separate the alkapton acid from the brown syrupy material with which it is mixed. The watery solution so obtained gave a dark blue reaction with ferric chloride, and on evaporation at a low temperature left a very scanty crystalline residue which was purified to some extent by re-crystallisation from ether. The crystals so obtained, although by no means satisfactorily pure, did not melt below 140°, whereas the melting point of uroleucic acid is 130° to 133° C., and a mixture of the two acids should have a still lower melting point. Hence it was concluded that the urine of Thomas P— did not contain uroleucic acid in any considerable quantity, or that if this acid were present it was only in very minute amount as compared with homogentisinic acid, and, indeed in quantities too minute to be detected by the methods employed in such small volumes of urine as were here available. The small ultimate residue apparently consisted of homogentisinic acid which has escaped precipitation as the lead salt.

Case 2.—The patient is a school-boy, aged 14, in good health, who has been alkaptonuric from infancy. He is a brother of the patient whose case was investigated by Drs. Armstrong and Walter Smith, of Dublin, in 1882, of whom the latter arrived at the conclusion that the abnormal urinary constituent was probably protocatechuic acid. The birth of the present patient and the fact that he had alkaptonuria were mentioned by Dr. Kirk in one of his papers.

By the kindness of Dr. C. E. Baker I was supplied with some three litres of the urine, but have had no opportunity of making any quantitative estimations of the excretion on successive days.

The urine had all the characteristic features already described, was acid in reaction, and had a specific gravity of 1025. On standing, even without the addition of an acid, deep brown crystals of uric acid were deposited. As estimated by Baumann's silver process,

10 c.c. of the urine had a reducing power equivalent to that of 0·0363 grm. of homogentisinic acid, *i. e.* about half the reducing power of that of Thomas P—.

By Wolkow and Baumann's method crystalline lead homogentisinate was readily obtained.

The urine also yielded crystals of this salt by the simple method above described, the yield being about half that obtained in the previous case.

The following figures were obtained with the product of the simple process, and they not only afford conclusive proof that here again one was dealing with lead homogentisinate, but they also show that the product obtained by my process is no less pure than that extracted by the process of Wolkow and Baumann :—

0·3127 grm. of the salt lost at 100° C. 0·0288 grm. of water of crystallisation = 9·21 per cent. (calculated = 9·08 per cent.)

0·2710 grm. of the dehydrated crystals yielded 0·1510 grm. of lead sulphate, equivalent to 38·05 per cent. of lead (calculated = 38·25 per cent.).

No special examination of free homogentisinic acid from the lead salt extracted from this urine was carried out, but a specimen obtained from a mixture of the lead salts from this and the previous case, in each instance extracted by my method, melted between 146° and 147°.

The mother liquor from which the crystals had been deposited (simple process) was treated as in the previous case, viz. by the addition of sulphuric acid, filtration from the precipitate of lead sulphate, and repeated extraction with ether. In the residue left after distilling off the ether a considerable mass of crystals of free acid formed, and these were transferred to a porous tile, which absorbed nearly all the coloured syrup and left the crystals nearly pure. After drying over sulphuric acid a specimen melted at 143° C., thus behaving as a slightly impure specimen of homogentisinic acid.

Seeing that uroleucic acid, if present in the urine,

should have constituted at least a large part of this scanty final product, and should have manifested its presence by a conspicuous lowering of the melting point, it seemed evident that this acid was not present in this urine in any appreciable amount.

CASE 3.—The specimens of urine which Dr. Pavy was kind enough to hand over to me for examination were passed by three members of the family already referred to. They had been standing in bottles for no less than eight years, were alkaline in reaction, and were for the most part almost black in colour. It was evident that in the lapse of time a considerable part of the alkapton acid originally present in them had undergone oxidation. However, the examination of these specimens promised to be of considerable interest, as one hoped that it might still be possible to detect the presence of homogentisinic acid in them.

The first specimen was passed by a youth aged 18 (Case 31 in the Table) ; 300 c.c. of the urine were available, consisting of a darker and a paler portion. These were mixed together, filtered from a bulky black sediment which had formed, and treated according to Wolkow and Baumann's process.

The addition of sulphuric acid caused much effervescence. From the aqueous solution of the residue from the ethereal extracts a crop of microscopic acicular crystals was deposited, apparently consisting of lead homogentisinate. The solution itself showed a transitory dark blue colour with ferric chloride. 0·2349 grm. of the crystalline deposit lost, at 100° C., 0·0152 grm. of water of crystallisation = 9·04 per cent., which agrees closely with the calculated figure, 9·08 per cent. 0·3042 grm. of the dried lead salt yielded 0·1718 grm. of lead sulphate = 38·57 per cent. lead. This too high percentage was probably due to incomplete removal of lead acetate. The estimation could not be repeated for lack of material.

It was, however, evident that, although so long kept, the urine still contained homogentisinic acid.

Case 4.—This was another of Dr. Pavy's cases (No. 30 in the Table). About 300 c.c. of this urine, which also consisted of a darker and paler portion and was alkaline in reaction, yielded by Wolkow and Baumann's method a fair crop of crystals, which had all the appearance of those of lead homogentisinate. The mother liquor yielded a transitory dark blue colour with ferric chloride.

0·3085 grm. of the crystals lost, at 100° C., 0·0282 grm. of water of crystallisation = 9·14 per cent.

The lead salts from both this and the previous specimen melted, with complete blackening, at about 215° C., which is given as the melting point of lead homogentisinate, but the exact temperature at which melting occurs is very difficult to determine, and the change which takes place at about 215° appears to be rather of the nature of a decomposition than simple fusion.

Here also the presence of homogentisinic acid was beyond doubt.

Case 5.—The greater part of the specimen from this case (No. 29 in the Table) was unfortunately lost, and although from the small amount remaining a small quantity of crystalline lead salt was obtained, this did not suffice for any satisfactory confirmatory tests, and therefore no safe conclusions can be drawn from the incomplete examination, although the appearance of the crystalline deposit left very little doubt that here also homogentisinic acid was present.

Leaving aside this fifth case, I am thus able to add four fresh cases of alkaptonuria to the list of those in which homogentisinic acid has been found in the urine.

These observations lend additional support to the view that homogentisinic acid is a constant constituent of

alkapton urines, and plays the chief part in the production of alkaptonuria. It has indeed been found in every case in which it has been specially looked for, and even in some of the cases examined before the publication of Wolkow and Baumann's researches re-examination of the material has led to its detection.

On the other hand, in two of the cases here dealt with, I endeavoured to ascertain whether uroleucic acid was also present, in both instances with negative results. Wolkow and Baumann equally failed to find any other reducing substance than homogentisinic acid in the urine of their patient, but in many of the cases since recorded no special search for uroleucic acid would appear to have been carried out.

It seems probable that uroleucic acid is rather of the nature of a bye-product, and that the urines examined by Kirk were peculiar in containing considerable quantities of this substance in addition to homogentisinic acid which, as Huppert has shown, was present in them in even larger amount.

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SERIES I.—*Published Cases.*

Date of publication and number	Sex	Age at time of observation	Duration of alkaptonuria	Family history	Associated maladies, &c.	Results of chemical examination of urine.	Observer.
1859 1	M.	44	Not stated	Not mentioned	Carcinoma of vertebral column	"Alkapton." Sugar also present	Bödeker
1875 2	M.	29	"	"	Phthisis pulmonalis	Alkapton	Fürbringer
1875 3	M.	4-15 months	First noticed in second week of life	"	Noticed after icterus neonatorum	Pyrocatechin	Ebstein and Müller
1875 4	M.	—	Not stated	"	Fractured rib and other injuries	"	Fleischer
1882 5	F.	3 years	Life-long	Sister of No. 26	—	Protocatechuic acid	Armstrong. Walter Smith
1884 6	F.	27	Noticed at age of 27	Not mentioned	Gastric ulcer two years previously	Probably protocatechuic acid	R. Maguire
1886 7	M.	8	Life-long	Brother of Nos. 8 and 9. An older brother, aged 10, not alkaptonuric	Healthy	From the urine of these patients Kirk isolated two acids, which he named uroleucic and uroxanthic respectively.	B. Kirk
1886 8	M.	—	"	Brother of Nos. 7 and 9	Died at age of three years of whooping cough	Huppert extracted homogentisinic and uroleucic acids from Kirk's material (1897)	"
1886 9	M.	Infant	"	Brother of Nos. 7 and 8	Healthy	—	"
1887 10	M.	—	Not stated	Brother of No. 22	Insurance case	Glycosuric acid — homogentisinic acid found by Baumann (quoted by Embden). Probably uroleucic acid, also (Huppert)	John Marshall
1886-7 11	M.	Young	"	Not mentioned	"	No protocatechuic acid—a crystalline acid obtained	Barton-Bruce

	Sex	Age				Homogentisinic acid demonstrated in this case	First in this case
1891 12	M.	68	Life-long	Brother of No. 13	Carcinoma of pros-tate	Homogentisinic acid demonstrated in this case	Baumann and Kraske. Wolkow and Baumann, Embden
1892 13	F.	60	"	Sister of No. 12	Morbus cordis	Homogentisinic acid	
1892 14	M.	—	Observed on a single day—recurred later (Embden)	Not mentioned	Diabetes	Glycosuric acid. The analytical figures agree with those of homogentisinic acid. Sugar also present	A. Geyger
1892 15	M.	—	Long standing	"	Regarded as diabetes	Homogentisinic acid, not sugar	Garnier and Voirin
1895 16	F.	—	Appearance concurrent with aggravation of malady	"	Pyonephrosis—operation—death	Homogentisinic acid	A. Slosse
1895 17	M.	45	Unknown	No other known case in family	Healthy	"	H. V. Ogden
1896 18	M.	18	Life-long, but apparently intermittent	No other case in family	Herpes of p uls. Dy-suria	"	P. Stange
1896 19	F.	43	Apparently as a pre-mortal symptom	Not mentioned	Phthisis and tubercular peritonitis	"	W. von Moraczewski
1897 20	M.	About 50	Probably long standing	"	Had previously suffered from sciatica and facial neuralgia	"	G. Denigès
1897 21	F.	17	For 3 days only during an attack of gastro-intestinal catarrh	No other case in family	Gastro-intestinal catarrh. No previous illness, except measles	"	C. Hirsch
1898 22	M.	57	Not stated	Brother of No. 10	Insurance case	Not yet published	T. B. Futcher
1898 23	M.	8	Life long	No other case in family, only brother, aged 9, not alcaptonuric	Healthy	Homogentisinic acid	Ewald Stäer
1898 24	F.	47	Life long	No other case in family	Inguinal hernia — operation.	"	Noccioli and Domentei

Series II.—*Unpublished Cases.*

Number.	Sex	Age at time of observation	Duration of alkaptonuria.	Family history.	Associated maladies, &c.	Results of chemical examination of urine.	Observer.
25	M.	3 months onwards	Noticed on second day of life	No known case in family — brothers and sister not alkaptonuric	Healthy. Influenzation of lungs at 2 months; diarrhœa at 10 months	Homogentisinic acid; uro-leucic acid not found	Dr. Voelcker's case.
26	M.	—	Life-long	Brother of No. 5. 3rd child	Healthy	,,	Dr. Baker's case.
27	M.	30	,,	No known case in family	Sacro-iliac and lumbar pain; floating kidney	—	Dr. Pavy's case.
28	F.	30	,,	9th of 14 children. Sister of Nos. 29, 30, and 31	Healthy	Homogentisinic acid—proof incomplete	,,
29	M.	—	,,	11th child. Brother of Nos. 28, 30, and 31	Healthy	—	,,
30	M.	22	,,	13th child. Brother of Nos. 28, 29, and 31	Urine supposed to contain sugar	Homogentisinic acid	,,
31	M.	18	,,	14th child. Brother of Nos. 28, 29, and 30	Refused at an Insurance Office as glycosuric	,,	,,

THE RESULTS

OF

SEGREGATION OF CASES AND MOVING FROM INFECTED SITES

IN

ERADICATING THE ASSAM EPIDEMIC MALARIAL FEVER OR KALA-AZAR

BY

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IN a paper that I had the honour of reading before this Society in March, 1898, I gave a summary of my investigation of the Assam epidemic malarial fever, locally known under the name of kala-azar, which I concluded to be an intensified form of malarial fever, which, through a succession of very unhealthy years due to extraordinary seasonal conditions, had attained the power of being slowly communicable, usually in an indirect manner, from person to person. I propose in the present paper to give the results of my recommendations for the eradication of the disease, which were based on the above

view, as the signal success which has attended them lends considerable support to my conclusions.

These recommendations—which were omitted from my previous paper for want of space—were mainly as follows:

(1) All newly imported coolies should be placed in fresh lines, and not allowed to enter old infected ones.

(2) In slightly infected lines all infected persons, together with their households, should be moved out into separate segregation lines, and the huts which they have inhabited should be burnt and not rebuilt.

(3) In badly infected lines all the healthy people should be moved out during the dry cold weather months, when infection is at a minimum, and placed in new lines, while the infected persons and their households should be segregated, and the infected line abandoned.

The results which have followed the adoption of these measures on various infected tea gardens in Assam will be briefly considered under the above headings:

(1) It was pointed out in my previous paper that this epidemic fever generally lasts for some ten years in any one district, or about six in any given village, and then dies out, passing on to fresh areas. There was, however, reason for believing that it would continue almost indefinitely in the case of coolie lines on tea gardens, as fresh subjects would be introduced year by year to replace those who had succumbed to the disease. The importance, then, of placing all newly arrived coolies in fresh uninfected lines was obvious, and the instances in which several hundreds of newly imported coolies have been so isolated in separate lines, which need not be more than a few hundred yards from the old infected ones, without one of them contracting the disease in periods of a year and upwards, have already been recorded in a note in the ' British Medical Journal ' of September 24th, 1898. The success of this measure is then proved, and need not be further dwelt on here.

(2) It will have been noted that under this heading it

was advised that not only those who were found to be suffering from the disease were to be removed from slightly infected lines, but also their households. The reason of this is that by the time a person, who is suffering from this form of malarial fever, has reached the stage of the affection when it is first possible to differentiate it from the ordinary non-communicable malarial fevers of Assam, he will have had fever for one or two months, and others, who are living in the same house, will be very likely to have already become infected. If they, too, are not removed a source of infection will remain, and the disease will not be controlled, much in the same way as used to be the case with regard to glanders before the introduction of mallein enabled those horses to be picked out who had already become infected, but did not yet show any clinical symptoms of the disease, yet were able to keep up the disease in a stable in spite of all clinically infected animals being slaughtered.

This measure has recently been carried out by Dr. Lavertine of the Nowgong district with the following results : In three coolie lines, the total inhabitants of which numbered over a thousand souls, seventy-three cases of kala-azar were found in May 1898, and, together with twenty-seven relatives, were removed and segregated at that time. In the next four months, including the most malarious season of the year, no new cases of the disease appeared in the previously infected lines, and at the end of November, when the fever season was quite over, only six cases of the disease had occurred in these lines since the removal of the infected people, four of these having occurred in one line from which more than half the infected people had come, and which was described as having been " formerly the hotbed of the disease ; " two in another line, while the third line remained absolutely free from the affection. When it is remembered that at least one of these lines was so much affected that it was a question whether it would have eventually to be abandoned altogether, and that the season when the infected

people were removed was later than was advisable, I quite agree with the opinion of Dr. Lavertine, who writes : " I look on the measure as an almost complete success. If this had not been done I doubt if we would have had less than 100 new cases." In this instance the segregation line was about three quarters of a mile from the old lines. It may also be mentioned that thirty-five of the segregated cases had died at the time of the report, and that the rest were still under observation.

(3) The third and most severe measure is that of clearing out and ultimately abandoning badly-infected lines. The following instance in which it was carried out by Dr. Dodds Price, also of the Nowgong district, on a tea garden which had previously lost over 200 coolies from the epidemic, is of great interest and importance. The line which was dealt with was so badly infected that early in 1897, that is at the time of the year when the epidemic is at its lowest annual ebb, out of 240 souls no less than 146 either were suffering from the disease or had cases in their households. The unaffected households, numbering 94 people, were moved into a new line, the general surroundings of which were precisely similar to those of the old one, and over 300 new coolies were placed in the same line. Shortly after they were moved there five cases of suspicious fever were noticed among the 94 people from the infected lines, and these five were immediately sent back, and two of them subsequently died of the disease. They had evidently contracted the fever before they were moved from the old lines, and as a consequence of this prompt action there has not been one death from kala-azar in these new lines in a period of twenty months, including two fever seasons. On the other hand, out of the infected people nearly one third had died in the same period, while what is still more conclusive, the disease spread to another small line containing sixty time-expired coolies adjoining the old infected line, and as they were not under contract and refused to be moved, one third of them died within fifteen

months. Moreover, in another infected line on this garden, which was at first not dealt with as above, the disease continued to prevail much the same as before, until the worst part of it was burnt down and abandoned, since which the disease has also decreased to a large extent here too. Dr. Price, in consequence of the above results, which contrast very favourably with his former disheartening experience in trying to combat the disease for several years before my investigation, now writes, " I say, unhesitatingly, no half measures. Burn down the lines, alter the site, and place none but non-infected people in the new lines. By so doing, thousands of rupees will be saved on large gardens, to say nothing of the saving in human life."

The above results will be sufficient to show that the measures that I advocated have been successful in actual practice, and as far as tea gardens are concerned,—and unfortunately it is only on them that these measures appear to have been as yet actively carried out—and this fearful epidemic disease, which carries off from 70 to 90 per cent. of those attacked by it, after a lingering illness lasting usually over six months, has now been largely robbed of its terrors, and brought within the pale of controllable epidemic diseases.

THE

REMOTE PROGNOSIS OF PERICARDITIS

BY

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(Communicated by Dr. FRANCIS WARNER)

The immediate prognosis of pericarditis has been satisfactorily worked out. It is well known that the rheumatic cases, as a rule, terminate favourably, while those associated with renal disease commonly, though not invariably, end fatally. The remote prognosis, though it has from time to time received attention from clinical workers, appears still to be a matter of doubt. I was, therefore, led to undertake an inquiry into the after-history of a large number of patients who had suffered from pericarditis, to determine, if possible, the factors which influence the ultimate prognosis in any individual case.

Previous Work.

It has long been recognised that considerable fluid effusions into the pericardial sac may be entirely absorbed,

but the possibility of the removal of fibrinous exudation without the formation of adhesions is still doubtful. It is certain that adhesions of greater or less extent occur in the majority of cases, and though there is no evidence that complete absorption does ever take place (1), the adhesions may be limited to a small area about the base of the heart, or to a single band between the parietal and visceral layers of the pericardium.

Adhesions being the common result of inflammation of the pericardium, it remained to determine how far they affected the comfort and well-being of the patient, and especially if they tended to shorten life. The earlier writers, Lancisi, Vieussens, and Morgagni (2), agreed that adherent pericardium was incompatible with long life. The same opinion was held by Corvisart (3). Laennec (4), on the other hand, was impressed with the fact that he had found in the post-mortem room universal adhesions in cases in which there was no evidence of cardio-vascular disturbance during life, and was led to conclude that such a termination of pericarditis was not unfavourable. Hope (5) followed the views of Morgagni and Corvisart, believing that, although for a time pericardial adhesions might not cause much inconvenience, the action of the heart in such cases was a "constant struggle," and that a fatal termination was inevitable. He believed that this occurred more rapidly in patients among the working classes.

Gairdner (6), in his classical paper upon the favourable terminations of pericarditis, pointed out that Hope's conclusions were too sweeping, and recognised that many patients, by the adoption of a "reduced scale of existence," might live in comfort for years.

Kennedy (7) analysed ninety cases of adherent pericardium in which there was no valvular disease, and found that hypertrophy and dilatation were a common, and atrophy an occasional result of pericardial adhesions. He concluded, therefore, that a most guarded prognosis should be given, and that in no case was it justifiable to

effect an insurance upon the life of the patient. Gairdner (8) wrote a strong criticism upon this paper, summing up his arguments thus :—" A certainly not small proportion of persons affected with pericarditis and its sequela of adhesion may survive the attack for many years, and may live in tolerable comfort, without the inconvenience attendant upon a dilated, hypertrophied, or atrophied heart."

Bauer (9) drew attention to the fact that in those cases in which the adhesions were the firmest, and the resistance to the movements of the heart presumably greatest, hypertrophy did not develop. This observation led many writers to put down hypertrophy in all cases of adherent pericardium to associated valvular disease (10), in spite of the fact that in Kennedy's ninety cases the valves were unaffected, and that Wilks (11) had shown that all the phenomena of failure of the heart from valvular disease may be produced by the influence of pericardial adhesions alone.

Sturges (12) and others, who have especially studied the cardiac diseases of children, have shown that the outlook in young subjects is decidedly unfavourable, inasmuch as the pericarditis is a part of a general carditis, the involvement of the myocardium being the most important factor. This opinion was supported by Dr. Lees and others in an interesting discussion at the Annual Meeting of the British Medical Association in 1898 (13).

In his monograph upon adherent pericardium (14), Dr. J. H. Broadbent states that " it is only in cases in which the heart is of normal size, and the pericardium is not adherent to the chest wall or to a large area of the diaphragm, that adherent pericardium gives rise to no serious consequences." Dr. Frederick Roberts (15) is inclined to agree with this, and believes that " there is a general tendency to make too light of the conditions remaining after acute pericarditis."

In a paper on the relation of heart disease to life assurance, Sir William Gairdner brought up the question

of the remote prognosis of pericarditis, and while recognising that pericardial adhesion is undoubtedly a lesion which would render a life insecure, Sir William is still in doubt whether acute pericarditis must necessarily end in adhesion (16).

A survey of the previous clinical work upon the subject leads, therefore, to the following conclusions :

(1) Pericarditis commonly results in adhesions.

(2) In some cases adherent pericardium is perfectly compatible with health.

(3) The size of the heart appears to be an important point. If large, the outlook is unfavourable.

(4) Hypertrophy does not appear to be due to the resistance imposed by the adhesions to the contractions of the heart.

(5) Myocarditis and valvular lesions are of greater import than the condition of the pericardium.

(6) Pericarditis in children is usually part of a general carditis, and the involvement of the myocardium in the inflammatory process is believed to be the essential feature in the rapid failure of the organ met with in the young.

RECENT PHYSIOLOGICAL WORK.

Recent physiological work has thrown much light upon the functions of the pericardium in animals, and the results of experiment appear to be so important that I will consider them before describing my own clinical work.

The pericardium consists of two essentially different parts, a serous sac enclosed in a tough fibrous bag. If a cannula be tied into the pericardium of a cadaver, and be then connected with a vessel of water raised to a height, the sac gradually dilates until the fibrous bag is tense. From this point onwards, even if the pressure is raised until it is equal to two atmospheres, no appreciable change takes place. Mr. Barnard (17) has shown that normally this inextensile sac prevents the heart dilating beyond a certain

point, and thus the mechanical disadvantages of dilated cavities and a thinned wall are prevented. For, in the first place, the heart will contain more blood when the fibrous pericardium is removed than when the organ is in the closed sac; and secondly, in conditions of extreme dilatation the heart actually "herniated" through an opening made in the pericardium, particularly in diastole. Pressure upon the abdomen by driving the blood from the great veins into the right heart produced the same result. Barnard further found that the extra blood was contained in the right heart, and that when the pericardium no longer supported the right ventricle, relative incompetence of the tricuspid valve occurred. Thus, in the light of these experiments, the pericardium may be considered to be to the heart what the adventitious coat is to an artery. Dr. Leonard Hill (18) has also shown some striking experiments which illustrate the same points. Animals with powerful abdominal muscles have a tough pericardium, whilst, on the other hand, those with weak abdominal muscles have a weak pericardium. He took for comparison the eel and the snake. The snake has a weak abdominal wall, and propels itself by muscles attached to its ribs, while the eel propels itself by its strong abdominal muscles. The pericardium of the snake is a weak bag, while the heart of the eel is contained in a very tough fibrous sac. If the eel be held up by its tail, blood will gravitate from the great abdominal sinuses into the heart, but no over-distension occurs if the pericardium be intact. If, however, the pericardium be open, the effect of gravity over-distends the heart, and the organ may be dilated to three times its normal size. Dr. Hill further showed that if it were not for the support of the pericardium, the muscular contractions of the abdominal wall would continually over-distend the heart. The weak pericardium of the snake was shown to afford a very poor resistance to the dilatation of the heart as the result of gravity.

If we apply what experiment has taught us occurs in animals to the conditions which obtain in man, it will be

seen that this protective support of the pericardium comes into play during muscular effort. All strong muscular contractions drive the blood from the veins of the limbs into the right heart, and especially in strains involving the fixation of the chest (such as lifting and carrying weights) the powerful contractions of the abdominal muscles drive the blood from the large venous sinuses in the abdomen into the right heart and distend it. Under normal conditions the fibrous pericardium prevents this dilatation from going beyond a certain point. If the glottis be open there is only this fibrous bag to prevent over-distension. If, on the other hand, the glottis be closed, as is usual in great muscular effort, the outflow of blood from the right heart is impeded, and this would directly aid dilatation if it were not for the fact that the closure of the glottis raises the intra-thoracic pressure.

I conclude, therefore, that (1) the pericardium prevents dilatation of the heart beyond a certain point; (2) this support is particularly of value in preventing dilatation of the right heart during muscular effort.

The conclusions thus reached render a reconsideration of the remote effects of pericarditis, and especially of adherent pericardium, necessary.

METHOD ADOPTED IN MY INQUIRY.

While Medical Registrar at the London Hospital I took 200 cases of acute pericarditis occurring during the years 1890 to 1897 inclusive, and endeavoured to trace them with the object of ascertaining the after-history, and I have been successful in following up 130.

Twenty-four of the patients traced had died before the inquiry was begun. In most the fatal event had occurred in a hospital or infirmary, and in the majority I have been able to obtain details of the post-mortem appearances. A few died at home, and I have carefully investigated the cause of death, and all cases of doubt have been excluded.

Of the 106 other cases I have personally examined 100, often at frequent intervals; the remaining six are either abroad or in the country, and in them I have had to rely upon their written account of their condition.

Of the 100 cases I have had under my own observation, nineteen are now dead, and most have died at the London Hospital or at the North-Eastern Hospital for Children. Wherever possible, I was present at the autopsy, and as in the other cases I have availed myself of the kindness of the registrars and resident medical officers of other institutions in obtaining details of the post-mortem appearances.

In all the cases I have endeavoured to obtain as full a history as possible, and as many of the patients have been at one time or another in different hospitals, I have had to trespass upon the kindness of the officials of these institutions, and would here gratefully acknowledge the assistance they have afforded me.

For the opportunity of following up so large a number of cases I have to tender my grateful thanks to the Staff of the London Hospital, and to my colleagues at the North-Eastern Hospital for Children, who kindly placed their cases at my disposal. Those patients who were well enough came up to see me at the London Hospital, while others were seen in the wards or at their homes.

In every case the features of the original attack were examined by reference to the original notes. The subsequent history was inquired into. The conditions at the time of examination were noted and recorded on clinical charts, and in many cases I have had the advantage of radiography in verifying the areas determined by percussion.

As a result of this extensive experience I have been led to the conclusion that simple adhesion of the layers of the pericardium is of little moment, and that the dilatation of the fibrous pericardial sac, softened and sodden by inflammation, is of far greater importance. This dilatation is brought about from within. The heart

dilates and distends the yielding pericardium, which, while still inflamed, becomes adherent to the chest wall and diaphragm.

PERICARDIAL ADHESIONS.

It has long been a matter of surprise that the two layers of the pericardium may be completely adherent without any evidence, during life, of interference with the cardio-vascular mechanism. It is quite a common thing to meet with a universally adherent pericardium in the post-mortem room in a case in which the heart was supposed to be healthy. Probably the most remarkable instance of this condition is a heart in the museum of the University of Edinburgh. The pericardium is universally adherent, and there is so great a deposit of calcareous material that " except for about one cubic inch " over the apex the cardiac envelope is as "firm as the skull " (19). In this case there was no palpitation and no pain. It has also been observed that in the cases in which the adhesions are very firm there may be no hypertrophy of the heart (20). This I have verified in the post-mortem room on several occasions. The point is an important one, as it shows that the presence of tough adhesions does not induce hypertrophy by resisting the contractions of the heart.

In these cases I have found that the heart is not a large one, or, as I would rather put it, the pericardium is not dilated. There may, or may not, be adhesions between the chest wall and the diaphragm and the fibrous pericardium, but these are of little moment if the pericardium, and therefore the heart, are not much dilated.

On the other hand, there are cases in which the adhesions play an important part. The contracting bands of organised exudation may compress the great vessels, and especially the great veins. Wilks (21) first drew attention to these cases, and at a recent discussion at the

Medical Society of London (22) Sir William Broadbent and Dr. West mentioned instances in which the vena cava inferior had been completely occluded. I have not seen an instance of this compression by the contraction of adhesions, but in one case I saw the left auricular appendix so bound down that its cavity was almost obliterated. Grave disturbance of the vascular mechanism is inevitable under these circumstances.

The foregoing groups of cases of pericardial adhesions, however, form but a small proportion of the whole. In a large majority the pericardial sac is found, upon opening the chest, to occupy a large part of the thoracic cavity. The fibrous sac is adherent to the chest-wall and diaphragm, often over considerable areas. The two layers of the serous pericardium are firmly adherent to each other, and the cavities of the heart are dilated. The important point is to determine how this dilatation is brought about. It does not seem possible that with the heart in constant movement the cavities could be pulled open by contracting bands of organised exudation, and to say that adhesion takes place while the heart is dilated does not simplify the matter, for the dilated cavity must be in continual movement or the circulation would cease. As a matter of fact, it is not uncommon to find tags attached to both layers of the serous pericardium, and these tags are evidently fragments of adhesions broken by the constant movement of the heart; and at the apex, where the amplitude of movement is greatest, the adhesions may be pulled out into bands an inch or two in length.

The essential feature of these, the most important cases of adherent pericardium, is, as I shall shortly show, not the extent of the adhesions but the amount of dilatation. The whole difficulty of the problem is to determine how the dilatation is brought about.

Before leaving the consideration of pericardial adhesions mention must be made of the observations of Chevers (23), who pointed out that atrophy of the heart

was sometimes associated with adherent pericardium. Kennedy (24) found it in five of his ninety cases of adherent pericardium without valvular disease, and subsequent writers have supported his observations. The atrophy has been considered to be due to the strangulation of the heart by the adhesions. Sibson (25) does not give a single instance of atrophy of the heart in association with adherent pericardium, and in the records of the London Hospital for recent years I have failed to find one case. Atrophy of the heart appears to occur in wasting disease only.

Conclusions.

(1) In very rare cases adhesions may seriously interfere with the circulation by compressing the great veins.

(2) An orderly working of an undilated heart is quite compatible with very extensive pericardial adhesions.

(3) Adhesions of themselves do not produce dilatation of the heart.

(4) The essential point is not the presence of adhesions, but the amount of dilatation.

DILATATION OF THE PERICARDIUM.

We have already seen that the special function of the pericardium is to prevent over-distension of the heart during muscular effort. There is, however, no doubt that the sac will increase in size if the heart undergoes hypertrophy as the result of repeated strains, or of renal disease, or of a chronic valvular lesion. It is obvious that the sac which envelops a heart weighing 30 oz. must be larger than one which surrounds a normal organ.

My contention, however, is that when the pericardium is softened and sodden from acute inflammation, it does not oppose a limit to the dilatation of the heart, and that it is this loss of the normal support which is a marked feature of many cases of acute pericarditis, particularly in the young. In some of the patients I have watched,

the dilatation has been progressive, and manifest to the ordinary methods of examination from day to day. As the dilatation increases the tricuspid valve becomes incompetent, and pulsation of the liver and veins develops. In these cases I have found that the pericardium was dilated, that the heart and especially its right chambers, were dilated, but that the valvular lesions were insignificant. The acute inflammation had involved the fibrous and serous layers of the pericardium. There were loose adhesions between the outer surface of the sac and the chest-wall, and between the visceral and parietal parts of the serous coat, but there had been no time for the fibrous pericardium to become firmly adherent to the chest-wall and diaphragm. The dilatation of the softened sac was therefore progressive, there was no limit to the dilatation of the cavities of the heart, and compensation could not be established.

In the majority of the cases the process stops short of this. During the acute stage the dilatation of the pericardium goes on to a greater or less degree, and then the fibrous sac becomes adherent to the thoracic parietes. The adhesions become organised and ultimately are so firm and tough that a condition of permanently dilated adherent pericardium results. If the dilatation of the sac be extreme, and the sac become adherent, the heart may be dilated beyond the power of compensation. In the cases which I have personally observed, I have found that death occurs in from three to nine months after the original attack (see p. 435).

But, if the dilatation be not extreme, and adhesion take place, that is, if we have a condition of moderately dilated adherent pericardium, the thickened fibrous sac still acts as a support to the heart in muscular effort. But the conditions are abnormal, the heart is dilated and hypertrophied, and instead of being supported by a pericardium of normal size, it is in an unduly large envelope. During rest and freedom from muscular effort, there is no great call upon the heart, but with the resumption of

muscular activity, compensation which has been effected during the resting period, breaks down sooner or later. If the pericardium has not become sufficiently organised to resist the strains imposed upon it by the distension of the heart by muscular effort, coughing, &c., the softened sac may yield and the compensation established by the rest may break down at once. More often time has been allowed for the consolidation of the sac, and compensation is maintained temporarily and then breaks down. The failures may occur again and again, and compensation be restored, until finally, the failure is complete and death results. One of the patients I have had under observation for some time led a precarious existence for fourteen years with a moderately dilated adherent pericardium. Most cases, as I shall show later, succumb much sooner than this (see p. 424).

There still remain for consideration the cases in which there are no signs of failure of compensation, those cases, in fact, in which the post-mortem examination alone reveals disease of the cardiac envelope. The essential feature, as previous workers have pointed out (26), of the cases of adherent pericardium which do well, and present no symptoms, is the normal size of the heart. However great the amount of adhesion, there is no great increase in the size of the heart, if the supporting sac is undilated.

The pericardium may be acutely dilated with effusion to a remarkable extent. In Sir James Alderson's case, figured by Sibson (27), the effused fluid weighed three pounds and a quarter. But the plastic form of pericarditis, which is so common in children, also softens the pericardium. This I have been able to prove by injecting normal and inflamed pericardiums in the post-mortem room. The softening effect of inflammation upon fibrous tissue is more marked in children than in adults. Another point of importance is that pericarditis, particularly in children, is often latent, and sometimes recurrent. Last autumn I saw a boy of nine, who had been under medical treatment

for " influenza " for a fortnight. The parents were led to believe that the child was not very ill, but they had noticed that his mind wandered at night. I found a soft pericardial friction over the base of the heart, and a mitral systolic bruit. There had been very little complaint of pain in the chest, and articular symptoms were practically absent. It struck me at the time that but for the examination of the chest this boy might have been allowed to run about for some days longer with a softened pericardium, and that the sac must inevitably have dilated. I have no doubt that this is one way in which dilatation of the pericardium is brought about, but I think that the too early resumption of muscular effort after an acute attack, and before the fibrous sac has become consolidated, is the most potent cause of such a condition.

The movements of chorea deserve special consideration as a factor in dilating the pericardium, as the association of acute pericarditis with this disease is not infrequent. The movements are often very violent and of long duration, and they must tend seriously to strain a softened pericardium, especially as they are not accompanied by a rise in the intra-thoracic pressure which is brought about normally in great muscular efforts by closing the glottis. I came, therefore, to the following conclusions:

(1) A pericardium softened by inflammation offers little or no resistance to the dilatation of the heart, whereas the special function of the normal sac is to prevent over-distension of the organ.

(2) The dilatation of the pericardium may be progressive, and then compensation is never established.

(3) If the dilatation be extreme, compensation is impossible.

(4) The greater the dilatation, the more difficult it is for the heart to remain compensated.

(5) The formation of adhesions between the dilated fibrous pericardium and the chest wall and diaphragm constitutes a condition of permanently dilated adherent pericardium. Compensation may be brought about, but

as the conditions are abnormal there is always a tendency for it to fail.

(6) Dilatation of the pericardium without the formation of adhesions may bring about the same results.

(7) Extensive adhesions with the pericardium undilated do not interfere to any material extent with the orderly working of the heart.

The Effect of Concomitant Valvular Lesions.

We have already seen that the whole sequence of events associated with failure of compensation may be produced by pericardial adhesions alone. It is, however, more common to find that the valves are affected at the same time as the pericardium. The mitral orifice is the most often involved, but the valvular lesion is frequently of secondary importance ; and we know that such a lesion, even when it has begun in childhood, may be, and commonly is, perfectly compensated for years. It is now generally recognised that some of the cases of mitral stenosis met with in adolescents and adults are the result of endocarditis occurring in early life. It is years —often many years—before the compensation breaks down. On the other hand, the characteristic feature of the cases associated with pericarditis is the rapid failure of the heart. It must, nevertheless, be recognised that the association of valvular disease with dilatation and adhesion of the pericardium increases the gravity of the case, since the valvular lesion introduces another factor tending to dilatation of the cavities and impairment of compensation. If the pericardium be undilated, valvular disease does not appear to be of so much importance. One man of twenty-seven who had well-compensated mitral and aortic disease when attacked with pericarditis, presented no more hypertrophy when I saw him six years later than he had before the attack of pericarditis.

Conclusion.

The association of valvular disease with pericardial lesions, especially with dilatation and adhesion of the pericardium, enhances the difficulties of compensation.

MYOCARDITIS.

The rapid dilatation of the heart which is so frequently seen in the rheumatic heart disease of children has been considered, as I have already shown, to be due to a general carditis. The endocardial affection being of minor importance, and the condition of the pericardium as regards dilatation not having been considered, attention has been directed to the myocardium. So far as pathological observations go, there does not appear to me to be sufficient evidence to warrant the contention that the dilatation is entirely due to myocardial degeneration. Dr. Poynton analysed the post-mortem records of the Children's Hospital, Great Ormond Street, and found that out of 150 cases in which death was due to heart disease, myocardial changes were noted in 23. In 13 the muscle was described as being soft and pale; in 4 it was fatty; and in 6 it was tough and fibroid. In the records of 62 post-mortem examinations made during recent years at the London Hospital on cases at all ages in which there was evidence of old pericarditis, I have found that there was fibrosis or other marked evidence of myocarditis in six instances only.

It appeared to me to be more profitable to examine carefully the myocardium of patients dying with acute dilatation of the heart, with pericarditis. I have cut sections of three—two specimens from children and one from the heart of a lad of eighteen. In each I have failed to find evidence of myocarditis, except in the layer of muscle immediately below the serous coat. As a precaution, I have sent specimens to be examined by independent pathologists, who have confirmed my own observations.

As we have already seen, the essential feature of the cases of so-called "pan-carditis" is the marked dilatation of the heart, and the consequent failure of compensation. By analogy with other toxic processes—*e. g.* diphtheria and enteric fever—it is urged that the rheumatic poison particularly affects the heart muscle, and that fatty change in this part of the organ is the cause of the dilatation. It is even urged that the toxine has a particular affinity for the fibrous valvular ring. Fatty change in the myocardium is undoubtedly injurious, but does it cause extreme dilatation of the heart and failure of compensation? Pernicious anæmia is a toxic process in which fatty degeneration of the heart is found in a marked degree; but even when it is extreme the heart may act quite regularly. Again, we do not meet with the phenomena of dilatation and failure of compensation in typhoid fever and diphtheria; and these are both toxic processes, in which myocardial degeneration is common. The typhoid heart is, as Dr. Sansom (28) has shown, a small heart. Since the publication of his paper, the results which he obtained by percussion have been completely borne out by a series of radiographs taken at the London Hospital.

Balint (29) has recently shown that in animals the removal of even two cusps of the aortic valve, and the production of fatty degeneration of the heart muscle by the injection of phosphorus oil, does not cause failure of compensation.

Local dilatation of a cardiac cavity—that is, cardiac aneurysm—can be brought about during the systole of the cavity provided that there is local weakness of the heart wall. But general dilatation of a cavity cannot take place during its systole. If the muscle is weak, its contraction must be proportionately weak; and if the resistance cannot be overcome, the cavity simply does not empty. During the diastole the cavity immediately behind, provided that its muscle can overcome the resistance, throws its blood into the unemptied chamber; and if

the heart wall is softened by inflammation or fatty changes the cavity will dilate. Strong contractions of the limbs and abdominal muscles by driving venous blood into the right heart will act powerfully in the same direction. But it must be remembered that if the pericardium is intact—that is to say, inextensile and undilated—the heart muscle, however soft it may be, cannot dilate beyond what it probably does during any violent exercise.

If, however, the pericardium is dilated and the heart muscle softened by some degenerative process, moderate muscular efforts may produce a greater dilatation than the normal, but only so far as the dilated pericardium allows. Again, let us suppose that the pericardium is moderately dilated, and that the heart within it is hypertrophied and somewhat dilated, but the compensation is so adjusted that the cavities empty themselves, and there is no strain upon the pericardium. In such a case any condition of degeneration of the heart muscle may so weaken the walls that the cavities cannot empty, and compensation fails.

Conclusions.

1. The layer of muscle immediately below the pericardium is usually inflamed in pericarditis.

2. General myocarditis is rare.

3. Extensive fatty changes in the muscle of the heart are found in conditions in which dilatation and failure of compensation are unknown.

4. Experiment bears out clinical experience upon that point.

5. Dilatation of a softened cavity can only take place during diastole.

6. The dilatation is limited by the amount of dilatation of the pericardium.

7. Fatty degeneration of the myocardium is often the result, rather than the cause, of failure of compensation.

§ *The question of hypertrophy.*—Hope (30) believed that hypertrophy of the heart was an invariable result of pericarditis. Fagge (10), on the other hand, thought that hypertrophy was always due to concomitant valvular disease, previous or concurrent. Kennedy showed (7), however, that hypertrophy was present in 56 per cent. of cases of adherent pericardium uncomplicated with valvular disease, while Gairdner made it only 33 per cent. (8). It has been held that the hypertrophy of the heart secondary to pericardial adhesions is not real hypertrophy at all, but a thickening of the organ by "changes mostly fibroid," secondary to an interstitial myocarditis (31). On the grounds already stated I cannot agree with this view. In one of my cases the heart weighed 31½ oz., and there were no valvular lesions at all. The muscle was an excellent example of genuine hypertrophy.

If the heart dilates, hypertrophy must follow if the patient lives long enough, and if a softened pericardium permits greater dilatation than any other condition, we should expect to find the heaviest hearts in patients who had suffered from pericarditis. This, as I shall shortly show, is the case. Hypertrophy may occur very rapidly. Quite recently I made a post-mortem examination of the body of a boy aged eleven. He had been ill for three weeks with rheumatic pericarditis and chorea. The heart weighed 12 oz., double the normal weight at that age. There had been no previous illness, so that there is every probability that the increase of weight was brought about in three weeks. Dr. Goodhart (32) described a case the history of which suggested that the heart grew to a weight of 19 oz. in three or four weeks as the result of an attack of pericarditis.

From the post-mortem records of the London Hospital for recent years I have collected a consecutive series of sixty-two hearts affected with valvular disease, in which there was evidence of old pericarditis. In only four of these, *i. e.* about 6 per cent., was the weight of the heart less than 10 oz.

In the appended table the weights of these hearts are compared with the weights of a consecutive series of hearts affected with valvular disease, but with no evidence of antecedent disease of the pericardium.

TABLE I.

Weights.	Valvular disease.	
	With pericarditis.	No pericarditis.
Over 40 oz.	3	0
30—40 oz.	5	1
25—30 oz.	7	5
20—25 oz.	7	6
15—20 oz.	22	28
10—15 oz.	14	18
Under 10 oz.	4	4
	62	62

It will be seen that the effect of valvular disease, as might be expected, is to increase the weight of the heart. Pericardial complications tend still further to hypertrophy. Sibson's tables give similar results (33).

The weights of the four heaviest hearts and the associated conditions are appended.

1. 47 oz. Male aged 20. Universal adhesions. Mitral and aortic disease. Pericarditis aged thirteen.

2. 45 oz. Male aged 25. Universal adhesions. Mitral slightly stenosed. Aortic incompetent. Pericarditis aged seventeen.

3. 40 oz. Male aged 18. Universal adhesions. Mitral slightly stenosed. Aortic incompetent. Pericarditis aged six.

4. 38 oz. Female aged 51. Universal adhesions. Mitral and tricuspid incompetent. Aortic unaffected. Several attacks of rheumatism, the first aged fifteen.

The involvement of the aortic orifice in three cases no doubt played a considerable part in producing the

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[1] All cases of valvular disease, rheumatic or other, are included.

enormous hypertrophy. It does not explain it all, as I have been unable to find a single case in which such an extreme condition was the result of valvular disease alone. In the following case there was exceptional hypertrophy and no valvular disease.

Weight 31½ oz. Male aged 21. Universal adhesions. Valves unaffected. Pericarditis aged sixteen.

. A remarkable point is the enormous hypertrophy met with in some of the young patients. The following instances illustrate this.

1. Weight 32 oz. Female aged 16. Universal adhesions. Mitral admitted the tips of two fingers. Tricuspid and aortic incompetent. Pericarditis aged eleven.

2. Weight 28 oz. Female aged 17. Universal adhesions. Mitral stenosis. Aortic healthy. First attack aged thirteen.

3. Weight 27 oz. Female aged 17. Universal adhesions. Mitral admitted two fingers to second joint. Other valves healthy. Myocarditis with recent hæmorrhagic patch in wall of right ventricle. Pericarditis aged one, five, and seventeen.

4. Weight 22½ oz. Female aged 12. Universal adhesions. Mitral and tricuspid incompetent. Pericarditis aged eight.

In the four cases in which the weight of the heart was not above normal the conditions were as follows :

1. Weight 9½ oz. Male aged 20. Pericardium universally adherent. No valvular disease. Empyema. No rheumatism. Ill a year.

2. Weight 9 oz. Male aged 72. Slight adhesions very old. Mitral and aortic valves slightly thickened. Rheumatic fever aged six.

3. Weight 8½ oz. Male aged 30. Universal adhesions. Mitral stenosed. Cancer of the sigmoid. Acute rheumatism aged sixteen, nineteen, and twenty-six.

4. Weight 8 oz. Male aged 34. Universal adhesions. Valves unaffected. Phthisis, syphilis, lardaceous liver, &c. No rheumatism.

The Effect of Age and Sex upon the Prognosis of Pericarditis.

In order properly to understand the effect of age and sex upon the prognosis of pericarditis, it is necessary to ascertain the relative frequency of the disease at different ages in the two sexes. The ages at which the original attack occurred in the 130 cases which I have had under consideration, are set forth in the appended table. I have made five groups.

1. Young children, five years and under.
2. School age, six to fifteen.
3. Years of early labour (these are all hospital cases). sixteen to twenty-five.
4. Mature cases, twenty-six to thirty-five.
5. ,, over thirty-five.

Table II.

Age at first attack of pericarditis.	Males.	Females.
5 years and under	4	6
6—15	27	45
16—25	20	11
26—35	8	1
Over 35	7	1
	66	64

It will now be most convenient to classify these cases according to compensation.

1. Those cases in which compensation was never established. These are the cases in which the pericardial dilatation is extreme.

2. Those in which compensation was established at least once, but which tended to break down, many of the patients coming under treatment again and again. Moderately dilated pericardium.

3. The cases in which compensation remains good. Undilated pericardium.

TABLE III.

MALES.

Age at first attack of pericarditis.	Never compensated.	Precarious compensation.	Good compensation.	Total.
5 and under ...	2 ...	2 ...	0 =	4
6—15 ...	3 ...	22 ...	2 =	27
16—25 ...	2 ...	10 ...	8 =	20
26—35 ...	0 ...	0 ...	8 =	8
Over 35 ...	0 ...	2 ...	5 =	7
	7	36	23	66

FEMALES.

5 and under...	3 ...	3 ...	0 =	6
6—15 ...	3 ...	40 ...	2 =	45
16—25 ...	0 ...	6 ...	5 =	11
26—35 ...	0 ...	0 ...	1 =	1
Over 35 ...	0 ...	1 ...	0 =	1
	6	50	8	64

The cases in which compensation was never established lived from three to nine months after the original attack of pericarditis.

The precarious cases present all varieties; in some compensation was established once only, in others the heart failed, and recovered again and again. In some cases the failures were spread over as many as eight years (one case, already referred to, went on for fourteen years with repeated slight failures, but I have not included it here as I am only dealing with the patients who have had pericarditis between 1890 and 1897).

It will be noticed that the female cases more commonly present the phenomena of precarious compensation than the males, and especially between the ages six to fifteen.

If all my cases had been watched for as long as five years it would have been an easy matter to have calculated an average mortality for this group, but it will be readily understood that the majority of the precarious

cases which I have been able to personally observe have had the original attack during the last three years, and are still living. I am afraid that the percentage death-rate which follows is based upon too few cases to be of great value.

Of nine males who had pericarditis between the ages six to fifteen, and in whom compensation was precarious, the average length of life after the attack was three and one third years.

Of sixteen females, in whom the attack occurred between the same ages, the average length of life was two and one-fifth years.

At the other ages there are still fewer deaths, but so far as they go they give for the males an average length of life after the attack of three and a half years, and for the females an average of three years. But there are only eight deaths in all.

Between the ages of twenty-six to thirty-five there were three deaths due to intercurrent disease, viz. diabetes, phthisis, and hæmorrhage from gastric ulcer. One female, aged forty, died apparently from the cardiac condition. The fatal event occurred suddenly at home, and the evidence at the coroner's inquest gave heart disease as the cause.

The following tables are, I think, of greater value in elucidating some of the factors which are to be considered in the prognosis in respect of age and sex. I have tabulated the condition of the pericardium in 200 consecutive autopsies on 100 male and 100 female patients dying in the London Hospital from heart disease. They are taken consecutively from December 31st, 1897, back to 1894, and, of course, include many of the fatal cases of pericarditis which I have traced. All cases in which the valves were diseased as the result of rheumatism, chorea, ulcerative endocarditis, renal disease, or atheroma are included. The cases are arranged in quinquennial periods, and each case is represented in the columns by the age at death.

TABLE IV.—200 *consecutive cases* (100 *males and* 100 *females*) *dying in the London Hospital from heart disease, collected from the post-mortem registers,* 1894 *to* 1897, *inclusive.*

MALES.

Quinquennium.	No pericardial complications recorded. Age at death.	Acute pericarditis. Age at death.	Old pericarditis, adhesions, etc.[1] Age at death.
1—5	—	—	5
6—10	8	6, 9	8, 8, 7, 7, 9
11—15	15, 14	12	13, 14
16—20	16, 19, 16	17, 19, 18	20, 16, 18, 19
21—25	22, 25, 25	22, 23, 21	25, 22, 21, 25, 23
26—30	27, 27, 30, 30	—	27, 28
31—35	31, 32, 32, 33, 35, 35, 33	34, 33	31, 35
36—40	40, 39, 39, 36, 38, 37, 38, 38	37	40, 36, 38
41—45	43, 42, 41, 43, 43, 44, 41, 45, 44	42	44
46—50	46, 49, 50, 50	49	50
51—55	53, 53, 52, 52, 51	—	—
56—60	56, 60, 58, 60, 59, 56, 56, 58, 57	—	57
61—65	62, 63, 61	—	—
65—70	—	—	—
71—75	—	—	72
76—80	—	—	—

FEMALES.

Quinquennium.	No pericardial complications recorded. Age at death.	Acute pericarditis. Age at death.	Old pericarditis, adhesions, etc.[1] Age at death.
1—5	5	—	—
6—10	9, 7, 9	9	9
11—15	12, 13, 11, 12	—	14, 15, 15, 13, 13, 15, 15, 14
16—20	17, 18, 17, 19	19	19, 20, 17, 19
21—25	21, 22, 24, 24, 21, 24, 24	24	23, 21, 25
26—30	30, 29, 30	29, 30	27, 30
31—35	32, 33, 34, 33, 35, 35, 35, 32	—	32
36—40	39, 37, 40, 36, 36, 39	—	38
41—45	41, 42, 42, 45, 44, 42, 41, 41	44	44
46—50	50, 49, 50, 47, 49, 48, 49, 50, 49, 47	49	—
51—55	51, 54, 55, 54, 52, 51	52, 52	51, 51, 54
56—60	57	60	—
61—65	61, 63, 63	—	—
66—70	68	—	—
71—75	—	—	—
76—80	77	—	—

[1] White or milk patches are excluded.

In the first column are the cases in which the pericardium was healthy. In the second are the cases in which there was evidence of acute pericarditis at the post-mortem examination. In the third those in which there was evidence of old pericarditis, such as local or general adhesions. Simple white or milk patches are excluded.

By this arrangement the special ages at which pericardial adhesions are found in the post-mortem room are seen at a glance. It will be observed that patients with valvular disease uncomplicated with pericarditis die at a much later age than those who have suffered from pericarditis.

Coming now to an analysis of the cases in which there was evidence of old pericarditis, a marked difference is seen in the two sexes, a difference which is not accounted for by the average incidence of the acute disease. In part this appears to be due to the difference in the rapidity of the increase in the body-weight in boys and girls. From five to nine a boy and a girl grow at nearly the same rate. But between ten and fourteen the boy's body-weight increases much less quickly than does that of the girl. The girl reaches her maximum annual increase in the eleventh, twelfth, and thirteenth years. The boy has a minimum annual increase in the tenth and eleventh years, but after that steadily progresses till he reaches sixteen, when there is the maximal annual increase. It will be seen from my tables that between seven and nine several boys died, but that after that there are very few deaths until just after the period of maximal annual increase of body-weight, and the rapid growth together with early years of active labour strain a heart hampered with the results of pericarditis and it is very prone to fail.

DIAGRAM *comparing the annual increase of body-weight in school boys and girls between five and seventeen years of age. The vertical height in the columns represents the annual increase in pounds (constructed from Dr. Bowditch's tables). The male line is marked thus...... the female—.*
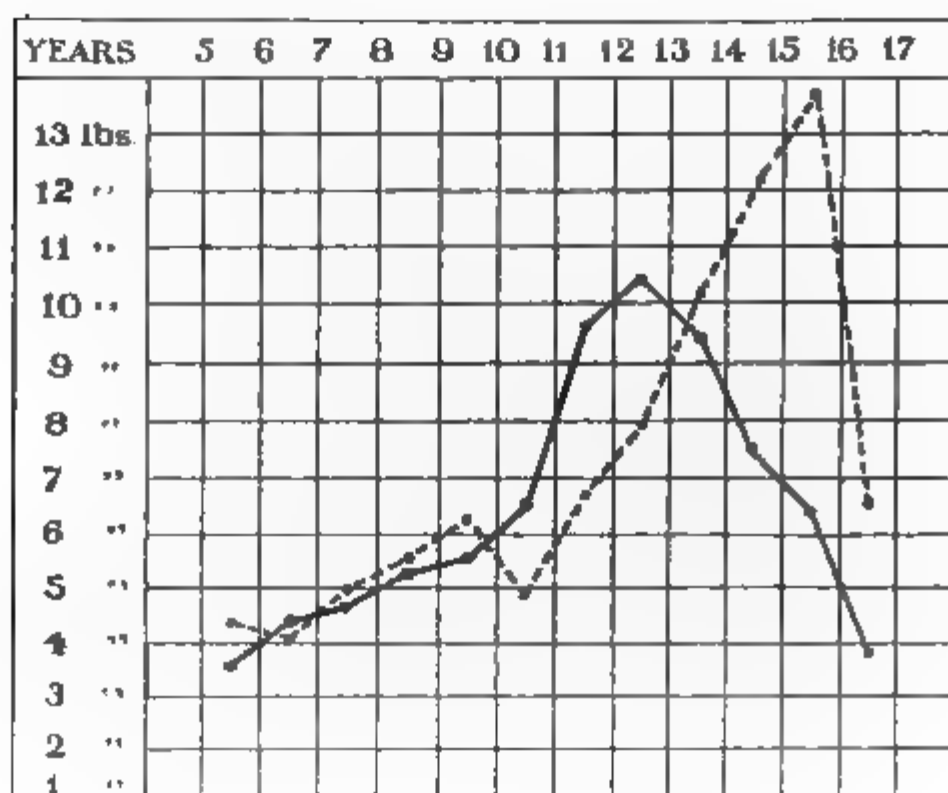
In girls the mortality is excessive between thirteen and fifteen, and is quite out of proportion to the number of cases, which itself is high. The most obvious explanation is the incidence of puberty, but the incidence of puberty is not accompanied by an increased death-rate in other affections. The essential feature is doubtless the rapid increase of the body-weight during this period.[1]

As these are hospital cases the influence of work may come in, for after leaving school some of the house-work involving lifting and carrying often falls upon these young girls. Later, between seventeen and thirty, the influences of hard work, pregnancy, and partu-

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[1] I am indebted to Dr. Francis Warner for calling my attention to the importance of considering the body-weight, and for references to Dr. Bowditch's statistics (34), from which the annexed diagram has been constructed.

rition are shown. It will be noticed that scattered through adult life and even in the senile cases evidence of old pericarditis is met with. Some of these are, no doubt, the well-compensated cases to which I have already referred. The male patient dying at the age of seventy-two had had rheumatic fever at the early age of six. I do not, however, think that we can assume that all of these patients suffered from pericarditis in early youth. In some the affection appeared to be due to extension from pleuro-pneumonia. In a few the adhesions found were probably the result of subacute attacks of pericarditis occurring in the course of renal disease. In three instances, in which there was no history of rheumatism, there were adhesions about the liver and spleen and evidence of syphilis, and I have been led to question whether some of these may not have been of syphilitic origin.

It will be noticed that the later deaths generally occur in the period of decay. The pericardial adhesions, if of early date, gave no trouble, and death was due to inter-current disease.

Conclusions.

1. In young subjects death occurs at different periods in the two sexes. In the females a pericardial lesion occurring in early childhood commonly leads to a fatal issue during or immediately after the period of most rapid increase of the body-weight, that is between the twelfth and fifteenth years. In boys there is a period of comparative immunity until the end of the years of rapid growth, that is until after sixteen.

2. Heavy labour, and in the females pregnancy and parturition, tend to cause failure of a heart hampered with the results of pericarditis.

3. The consideration of these influences points to some mechanical interference with the cardio-vascular mechanism, since it depends rather upon the growth of the body

and the muscular power exerted, than upon any other vital condition.

4. A certain proportion of the cases live until the period of decay, and these are, no doubt, the cases of undilated pericardium which have been already considered.

CLINICAL OBSERVATIONS.

Clinically, as I have already indicated, the cases fall into three great groups :

1. Those in which compensation is well established.
2. Those in which compensation easily breaks down.
3. Those in which compensation is never established.

1. *Well-compensated cases.*—Thirty-one out of my 130 cases come into this group. I have had frequent opportunities for observing nearly all of these patients. The youngest had the acute attack of pericarditis when fourteen years old. A reference to Table III will show that the male cases preponderate, and that adult males who suffer from acute pericarditis almost invariably do well. A careful survey of the histories has convinced me that any condition which necessitates a prolonged confinement to bed tends to a favourable issue ; that is, time is required to render compensation secure, or as I would prefer to put it, resumption of the round of daily exertion should not be undertaken until the risk of dilatation of a softened pericardium is past. One of the worst cases, a man of twenty-one, had articular rheumatism, endocarditis, pericarditis, and chorea. There were many subcutaneous nodules, and after the acute symptoms had subsided a series of relapses of the articular pains took place. The nature and multiplicity of the lesions kept this patient confined to bed for four months. I saw him over two years later ; he was perfectly well, and had had no symptoms. There was a systolic bruit at the apex, and that was all. A similar favourable result has been observed in several other cases, in which, for one reason or other, a long confinement to bed was necessary.

One important fact has struck me in the physical examination of all these cases : there is no great increase of the precordial dulness. Definite exocardial sounds were heard in five out of the thirty-one cases examined. As a rule they were present at the base of the heart. One lad of seventeen, who is engaged upon a sailing barge, complained of pain when he had to pull upon his ropes. He had a well-marked exocardial sound at the base, and located the pain in that region. In the other cases there was no complaint of dyspnœa or of pain, and in no single instance, even with the history before me, did I find evidence of old pericarditis beyond the sounds mentioned above. It is certain that some of these patients had pericardial adhesions, but I was unable to find definite evidence of them. As a matter of fact, these are the cases in which a diagnosis of pericardial adhesions is of little moment. The pericardium is undilated, the heart is undilated, and the adhesions give no trouble of themselves, although they would probably handicap the patient in the event of an acute illness supervening.

2. *Cases in which compensation easily breaks down.* —Eighty-six of the 130 cases come into this group. They are of all gradations, from the patient who has been in and out of hospital for years to one in whom compensation has been established but once.

The younger patients, and especially the young females, form the bulk of the cases, and a few of the patients in whom the original attack occurred after thirty-five must be included. (See Table III.)

Where there are so many gradations the signs and symptoms vary very much. In some cases it would be impossible in the absence of a history to arrive at a correct diagnosis, but if the condition be borne in mind I am certain that dilated adherent pericardium can be diagnosed with precision in not a few cases. The history is, of course, of great assistance, and if the case has been watched throughout there is very little risk in overlooking

it. It will be remembered that Sturges (12) found evidence of pericarditis in 94 per cent. of the children who die from heart disease, and a history of rheumatic heart disease in early youth, especially if followed by frequent failures of compensation, should at once lead to a careful search for evidence of adherent pericardium.

Symptoms and physical signs.—The aspect of the patient will vary somewhat with the age at which the acute attack of pericarditis occurred. If it was early there is often a peculiarly youthful appearance. Puberty is delayed, and in several instances I have seen lads and girls of seventeen looking no more than thirteen. The face is pale, or there may be a " mitral " flush upon the cheeks. In prolonged cases the fingers and toes are clubbed. There is emaciation and dyspnœa on exertion. Many, even of the less acute cases, cannot lie down at night. During the period of failure orthopnœa is a constant symptom. The presence of dropsy depends upon the condition of the compensation at the time, but it is remarkable how little dropsy there is in some of the young children. In them the great stress appears to fall upon the liver, which is greatly distended and tender.

Pain is a very prominent feature. The precordium is the chief seat, but it may radiate down the inner side of either arm and to the back. In several cases I found that it was referred to the left shoulder. These pains are the referred pains of the dilated heart, and are frequently accompanied with cutaneous hyperæsthesia. Both the pain and the hyperæsthesia are essentially different from the pain and tenderness of acute pericarditis, as Dr. Henry Head (35) has pointed out. They are evoked by lightly picking up the skin, and are diminished by firm pressure, while the pain in acute pericarditis is increased by pressure and by percussion. One of my own patients, a boy of eight, has shown these phenomena several times when he has come up for treatment. These referred pains involve the cardiac area pointed out by Dr. Head. They are essentially transitory, and often last but for a few

hours. In some cases the pain has a remarkably wide distribution. One case under my care had a band of hyperæsthesia extending from the nipples to the level of the umbilicus, and from the angles of the scapulæ to the loins. It will be understood that I do not regard these pains as in any way pathognomonic of dilated pericardium, but in that condition great dilatation of the heart is the rule, and, as Dr. Head has pointed out, a constantly dilated hollow viscus, which from time to time is subject to further distension, presents just the conditions which are likely to cause referred pains.

From time to time the cardiac distress becomes acute, and cyanosis and grave dyspnœa are manifest. Sometimes the attacks may be warded off by timely rest. One patient I have traced tells me that she has to spend about one day a week in bed. Even when the pericardium is very dilated, it is remarkable with what rapidity a few days' rest restores the easy working of the heart. One child of seven, in whom the cardiac dulness reaches from nipple to nipple, has been an in-patient in the London Hospital eight times in eighteen months. At last compensation cannot be restored, and death follows.

In young subjects the chest wall is bulged on the left side. The area of visible pulsation is often very extensive. I have seen it as high as the second left interspace and to the right of the sternum. The impulse often has a curiously rippling character in the intercostal spaces just above the apex. In many cases the action of the heart is obviously laboured. Retraction exactly at the apex during the systole is undoubtedly a valuable sign of adherent pericardium, but is by no means always present. In three instances I have seen systolic retraction of the lower intercostal spaces, pointed out by Dr. J. H. Broadbent as a characteristic sign of pericardial adhesions. Occasionally the epigastrium is retracted at each systole, but I have only observed it in two of my cases, and I have not seen the lower end of the sternum pulled in. On palpation a diastolic shock may be

frequently felt in the front of the chest, but in my experience very rarely posteriorly. The hypertrophied right ventricle is often felt pulsating in the subcostal angle.

On percussion the area of cardiac dulness is always increased, and corresponds with the dilated pericardium. The dulness may extend from nipple to nipple, and on the left side as far as the anterior axillary line. I have been struck with the fact that the margin of the dulness is often particularly well marked, especially along the left upper border. If it extends above the third rib, and if the dull area in the second left interspace is not altered by a deep inspiration, there is good ground for suspecting antecedent pericarditis, provided that retraction of the upper lobe of the left lung as the result of pleurisy or pulmonary disease is excluded.

I have found radiography of great assistance in verifying the results of percussion. In a few instances it has appeared to me that the fixation of the dilated cavities by adhesions has led to a more definite margin, especially along the left upper border. Examination with the fluorescent screen has been of value, but until there is greater steadiness in the illumination the difficulties are very great.

Auscultation does not give much assistance. A systolic bruit is almost always present. It is frequently heard over a very large area in front, and for some reason appears to be particularly well conducted to the back, being often heard at the angles of both scapulæ. It usually entirely replaces the first sound. A rough presystolic murmur is often present, even when there is no stenosis of the mitral orifice; and this fact, together with the frequent absence of the second sound at the apex, often leads to the diagnosis of mitral obstruction, although the sharp first sound characteristic of the latter condition is not heard. In one case which I observed there was such a murmur, and at the autopsy the mitral orifice was found to admit four fingers. Sometimes this bruit, which

must be of exocardial origin, is unaffected by pressure and by respiration. I am inclined to exclude mitral stenosis in very young children, even when a rough presystolic bruit is heard at the apex, because contraction of the mitral orifice is rare in the very young.

Exocardial sounds at the base of the heart may also lead to grave errors in diagnosis. In one case which I saw in 1897, although pericardial adhesions were diagnosed, the double bruit heard at the base was thought to be due to aortic stenosis and regurgitation. The heart was very large, and the hypertrophy and dilatation were considered to be mainly due to the valvular lesion. At the autopsy the pericardium was found to be universally adherent. It was hugely dilated, and the heart weighed thirty-one and a half ounces, but the valves were quite healthy. In several other cases I have found that aortic lesions have been diagnosed on account of the presence of exocardial sounds at the base of the heart.

During the period of failure the heart is always rapid and often irregular. One case was admitted to the hospital suffering from tachycardia. There were no bruits, and at the autopsy a dilated heart with adherent pericardium was found. The pulsus parodoxus has been shown to be by no means pathognomonic of adherent pericardium, and it is in my experience rare. The surface veins are commonly dilated, and those of the neck often show the phenomenon of diastolic emptying, frequently miscalled pulsating veins. The liver is distended and tender, and, as I have already remarked, the distension is greater in the young cases. When the dilatation of the right heart has led to tricuspid regurgitation there is pulsation of the liver and of the cervical veins. In those cases in which the inferior vena cava is compressed the ascites and oedema are sometimes indistinguishable from the conditions found in cirrhosis of the liver.

Many of the younger patients have a particularly good appetite and are free from gastric troubles for a long time, but ultimately the chronic congestion of the stomach

induces pain after food, flatulence, and vomiting. Towards
the end vomiting is persistent, but it does not then appear
to be altogether due to the condition of the stomach, but
rather resembles that found in association with renal disease
or toxæmia. In any case, severe vomiting is a grave
symptom. Back pressure upon the kidneys naturally re-
sults in a diminished flow of urine with the presence of
albumen.

Sleeplessness is a very troublesome symptom. In the
periods of cardiac failure the patient cannot sleep in the
horizontal position; and I have found that many patients,
even in the intervals, prefer to be propped up in bed.
The older patients often complain of dreams which cause
them to awake frightened. Occasionally there is great
mental excitement with hallucinations, particularly of
sight and hearing. The pain in some of the younger
patients causes grave nervous symptoms. In one infant,
aged fifteen months, the constant cry suggested menin-
gitis, and the case was sent to me as probably suffering
from that disease. In one case death was preceded by
twitchings of the right arm, leg, and face. There was
no evidence of uræmia, and at the autopsy there were no
obvious signs of disease of the brain. It is not at all
uncommon for the nervous symptoms to so mask the con-
dition, that attention is directed to the brain and not to
the pericardium and heart.

Manner of death.—Some of the patients died quite
suddenly, the heart being arrested in a condition of
asystole. In most cases the body becomes more and more
water-logged, and the failure is gradual. As I have
already said, death may be ushered in with fits, which
are not of uræmic origin. In one case, however, the
condition of the kidneys appeared to be the determining
factor in the production of puerperal eclampsia, the
patient dying from uræmic convulsions soon after the
birth of her first child.

3. *Cases in which compensation is never established.*—
Here the failure is progressive from the first. The

orthopnœa of the acute stage is persistent. Vomiting is early, and the nervous symptoms are marked. I have seen the precordial dulness steadily increase, and in one case it extended from the right nipple far into the left axilla. The dilatation in some instances has been so extreme that the heart has been punctured, under the impression that the enormous area of dulness could only be produced by a large pericardial effusion. The essential point is that it could only occur in a dilated pericardium. The heart becomes more and more feeble, and there is tricuspid regurgitation. The evidence of back pressure becomes more and more marked, and death occurs in from three to nine months after the acute attack.

Final Conclusions.

The remote prognosis of pericarditis depends in the main upon the amount of dilatation of the pericardium. The dilatation is due to the softening of the sac by inflammation. It may occur at once before the patient has recovered from the acute illness. It may be brought on by too early resumption of work after the acute attack, or it may be due to the patient keeping about while there is latent inflammation of the sac.

The dilatation may be progressive, and then compensation can never be established, but a similar result occurs if the primary dilatation be excessive.

The adhesion of a dilated pericardium to the chest-wall, while it renders the condition of dilatation permanent, may be of advantage when the adhesions become consolidated, for the thick sac acts as a support to the cardiac cavities. If the dilatation of the sac is not very great, the support thus afforded in some measure replaces that of the normal pericardium ; but as the cardiac envelope is dilated beyond its normal dimensions, so also is the heart. In these abnormal conditions there is a great tendency for the compensation to be impaired. Such

cases may live for years after the original attack; but they are subject to frequent breakdown, and are in a precarious state. If the pericardium is undilated there may be universal adhesions, but these are of no moment.

As regards age and sex the young cases show a greater tendency to dilatation of the pericardium, and therefore do worse than the older patients. The period during which there is rapid increase of the body weight is one which is especially trying to a heart working in a dilated pericardium. In girls who have suffered from pericarditis in childhood, and in whom compensation has been more or less established, the failure is almost certain to take place betwen the thirteenth and fifteenth years. In women the years of heavy work, pregnancy, and parturition are those in which failure of the heart is prone to occur.

In boys the failure occurs later than in girls—in the sixteenth and seventeenth years, or in the years of early labour. In adult males the prognosis is good, but heavy work, especially soon after an acute attack, leads to dilatation of the softened sac, and consequent failure of compensation.

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TABLE OF CASES.

TABLES OF CASES.

Cases in which Compensation was never established.

No.	Name.	Age.	Date of pericarditis.	Ætiology.	Date of death.	Remarks.
1	Harry P.	15 mos.	June, 1896	?	Sept. 21, 1896	Universal adhesions; heart large; valves unaffected.
2	Florence C.	4	Christmas, 1890	Rheumatism	June 29, 1891	Universal adhesions; heart 5 oz.; mitral slightly dilated.
3	Emily L.	5	June 3, 1891	,,	March 31, 1892	Mitral slightly stenosed; recent endocarditis; universal adhesions; heart 10 oz.
4	Sarah Ann R.	5	Oct. 27, 1893	,,	June 10, 1894	In hospital practically till death. M.S., bruit. No P.M.
5	Sidney D.	5	Early in 1896	,,	June 1, 1896	Universal adhesions; valves healthy.
6	Michael P.	6	April, 1898	,,	June 27, 1898	Huge dilatation; M.S. No P.M.
7	Chas. S.	8	Sept. 29, 1897	Chorea	Feb. 11, 1898	Universal adhesions; heart large; mitral two fingers; small vegetations on aortic.
8	Arthur M.	9	Dec. 11, 1895	Rheumatism	March 22, 1896	Universal adhesion; recent vegetations on mitral, but no stenosis; heart large.
9	Eliza H.	12	April 26, 1894	,,	Jan. 26, 1895	Universal adhesions; mitral stenosis; recent vegetations on mitral and aortic; heart 22 oz.
10	Rose C.	12	Aug. 7, 1894	,,	Sept. 30, 1894	Removed by friends and died at home. No P.M.
11	Lizzie B.	14	Dec., 1896	,,	June 30, 1897	Universal adhesions; recent vegetations on mitral and aortic; no stenosis; heart 19 oz.
12	Alfred S.	17	Sept., 1894	,,	March 4, 1895	Universal adhesions; mitral two fingers; aortic slightly incompetent; heart 19½ oz.
13	Thos. T.	21	June 26, 1895	,,	July 6, 1896	Complicated with ulcerative endocarditis; universal adhesions.

Cases in which Compensation is easily broken.

MALES.

No.	Name.	Age.	Date of pericarditis.	Etiology.	Latest information.	Remarks.
14	Charles K.	8	1890	Rheumatism	Seen May, 1899	Always ailing. Huge heart. Adhesions diagnosed. (See note at end of tables.)
15	David S.	13	1890	,,	Seen Sept. 31, 1898	Always under treatment. Several times warded. A.S., A.D., M.S.
16	Sidney R.	10	Jan. 28, 1890	Rheumatism, chorea	Died, L.H., May 10, 1891	Nodules. Readmitted with fresh pericarditis. P.M.—Adhesions; valves little affected.
17	Albert D.	12	1891	Rheumatism	Died, L.H., April 5, 1898	Several times in hospital. Double bruits at base (1898). P.M.—Adhesions; heart 15½ oz.; mitral affected; aortic free.
18	Morris R.	13	Dec. 16, 1891	,,	Died, L.H., May 21, 1897	Several times in hospital. Enormous heart, thought to be effusion; A.S., A.D., M.S. P.M.—Adhesions; heart 47 oz.; aortic and mitral affected.
19	John D.	16	1892	,,	Died, L.H., July 28, 1897	Often under treatment. Double bruits at base. P.M.—Adhesions; heart 31½ oz.; valves healthy.
20	Charles L.	7	1893	Chorea, rheumatism	Seen July 5, 1898	Cannot play because of pain at heart; A.C.D. not much enlarged; M.P.S.
21	Robert C.	11	1893	Chorea	Died, L.H., July 7, 1896	Several times in hospital. P.M.—Adhesions; large heart; mitral stenosis.
22	Alexander C.	17	1893	Rheumatism	Died, L.H., Jan. 29, 1897	Always ailing. P.M.—Adhesions; mitral incompetent.
23	Samuel G.	10	May 11, 1893	,,	Seen June 11, 1898	Well nourished; slightly cyanosed; A.C.D. increased; M.S.
24	George S.	10	June 30, 1893	,,	Seen July 5, 1898	No school; several times laid up; A.C.D. large; A.S., A.D., M.S.
25	Robert S.	18	Sept. 9, 1893	,,	Seen June 25, 1898	Anæmic; A.C.D. not much increased; M.S. and exo-cardial: veins distended.

No	Name.	Age	Date of pericarditis.	Ætiology.	Latest information.	Remarks.
26	William L.	4	1894	Rheumatism	Seen July, 1898	A.C.D. much increased; M.S.; signs of adhesions; pulsating veins, &c.; nodules.
27	Arthur R.	5	1894	,,	Died, L.H., Nov. 2, 1896	A.C.D. large; M.S.; nodules. P.M.—Adhesions; mitral slight stenosis; heart 13 oz.
28	Chas. D.	6	Feb. 3, 1894	,,	Died, L.H., Nov. 2, 1896	Large heart; M.S., M.P.S.; nodules. P.M.—Adhesions; mitral stenosis.
29	Chas. S.	13	June 2, 1894	,,	Seen July 5, 1898	Ill developed; anæmia; large heart; exocardial at base; M.S.
30	Thomas P.	12	Sept. 4, 1894	,,	Seen June 11, 1898	Anæmic; wasted: no school. Large heart; adhesions diagnosed.
31	Albert B.	17	Sept. 26, 1894	,,	Died at home, Dec. 26, 1898	Often in hospital. Adhesions diagnosed. No P.M.
32	Herbert J.	10	Oct. 9, 1894	,,	Seen June 22, 1838	Wizened, small boy; looks younger. Adhesions probable.
33	Gershon L.	10	Feb. 10, 1895	,,	Died, L.H., Sept. 28, 1898	Often under treatment. Large heart; M.S., bruit. No P.M.
34	Edward S.	15	April 9, 1895	,,	Seen July 5, 1898	Cannot work. In and out of hospital and infirmary. Large heart; M.S.
35	Henry H.	17	June 13, 1895	,,	Seen Nov. 9, 1898	Anæmic: large heart: A.S., A.D., M.S. Attends L.H. out-patient department.
36	William C.	37	Sept. 11, 1895	Extension	Seen June 18, 1898	"Short-winded," and not the man he was. A.C.D. large; M.S.
37	John C.	11	Sept. 30, 1895	Rheumatism	Died, L.H., Jan. 8, 1897	Adhesions diagnosed. Confirmed P.M. Mitral stenosed; large heart.
38	Arthur C.	16	Nov. 16, 1895	,,	Seen often, 1897–98	Adhesions diagnosed. Frequently in hospital.
39	Albert B.	17	Nov. 19, 1895	,,	Sister seen, June, 1898	Has had to give up work. Lives in country. Œdema of feet from time to time.
40	Clifton W.	8	Feb., 1896	,,	Seen March, 1899	No school. Always under treatment. Adhesions diagnosed. Final failure appears imminent.
41	Fred S.	15	Aug. 7, 1896	,,	Seen June 11, 1898	A clerk; very anæmic; wasted; dyspnœa; cardiac area large; M.S.
42	William S.	46	Oct. 17, 1896	,,	? Seen June 13,	Much dyspnœa; heart irregular; large dulness; no bruit;

43	William C.	6	Nov. 1, 1896	”	Died, L.H., July 26, 1897	Recurrent pericarditis. P.M.—Adhesions; mitral thickened; not stenosed.
44	William E.	22	Dec. 10, 1896	”	Seen Aug. 9, 1898	Large heart; A.S., A.D., M.S.; adhesions probable. Pain with heavy work.
45	Wm. H. B.	11	Jan. 4, 1897	”	Seen June 13, 1898	Thin, anæmic; heart area large; M.S.
46	Fred. Y.	23	Jan. 4, 1897	”	Seen May 22, 1898	Pain ever since. Anæmic; M.S., M.P.S.; double ? exo-cardial sounds at base.
47	Chas. S.	11	Feb., 1897, Great Ormond Street	”	Seen June 13, 1898	In hospital twice since. Nodules; large heart; adhesions probable.
48	Thomas W.	40	June 29, 1897	?	Seen June 13, 1898	Cannot work so well; dyspnœa; heart area large; M.S.
49	George D.	9	July 23, 1897	Rheumatism	Seen June 13, 1898	Small, weak, and anæmic; dyspnœa; large heart; M.P., M.P.S.
50	William C.	21	Sept. 20, 1897	”	Seen Nov. 10, 1898	Huge heart; adhesions diagnosed; A.S., A.D., M.S.

FEMALES.

51	Ellen C.	8	Jan., 1890	”	Seen Oct. 30, 1898	Anæmic; emaciated; adhesions; several times in hospital.
52	Catherine T.	13	July 1, 1890	”	Died, L.H., Aug. 7, 1894	Huge heart; several times in hospital; no P.M.
53	Alice W.	14	Oct. 23, 1890	”	Died, L.H., June 24, 1892	Huge heart; never well after attack. P.M.—Adhesions; heart 32 oz.; mitral stenosis.
54	E. A.	14	1890	”	Died, L.H., Sept. 8, 1895	Ill off and on; A.C.D. large. P.M.—Adhesions; heart 27 oz.; mitral incompetent.
55	Selina M.	13	Dec. 20, 1891	”	Died, L.H., Sept. 27, 1894	Several times in hospital. P.M.—Adhesions; large heart.
56	Frances S.	9	Jan. 6, 1892	”	Seen May, 1898; heard from Oct., 1898	Ill off and on ever since; small, anæmic; too ill to come up for examination. ? Since dead.
57	Kate W.	10	Jan. 28, 1892	”	Died at home, 1894	Several times in hospital; failing compensation.
58	Caroline D.	12	July 2, 1892		Died, L.H.	Several times in hospital. M.S., M.P., P.M.—Adhe...

No.	Name.	Age.	Date of pericarditis.	Ætiology.	Latest information.	Remarks.
59	Sarah B.	7	Oct. 3, 1892	Rheumatism	Died at home, Dec. 30, 1896	Never well since; has been at several hospitals.
60	Amy B.	15	1893	„	Died, L.H., Jan. 21, 1895	Several times in hospital; fresh pericarditis before death. P.M.—Adhesions; heart 19 oz.; valves normal.
61	Mary Ann I.	16	Feb. 1, 1893	„	Died, L.H., Aug. 22, 1898	In hospital in 1896; married 1897; 6 months pregnant on admission. Death from uræmia. P.M.—Adhesions local; dilated pericardium.
62	Maud M.	10	Aug. 9, 1893	„	Seen June 7, 1898	In hospital frequently; several subacute attacks.
63	Mary D.	12	Sept. 6, 1893	„	Died, L.H., Oct. 15, 1895	Double mitral and aortic bruits. P.M.—Adhesions; heart 17½ oz.; mitral incompetence; aortic free.
64	Elizabeth F.	11	Sept. 12, 1893	„	Seen July 1, 1898	A.C.D. not much increased; M.S.; anæmia; pain; œdema of legs.
65	Sarah A. K.	5	Oct. 27, 1893	„	Died, L.H., June 5, 1894	Went out compensated; returned in a week and rapidly failed. No P.M.
66	Elizabeth D.	14	1893	Scarlet fever	Seen June 11, 1898	Frequently in hospital; A.C.D. large.
67	Sarah B.	12	Feb. 20, 1894, E.L.C.H.	Rheumatism	Died, L.H., April 17, 1897	M.S, M.P.S., bruits. P.M.—Adhesions; heart 19½ oz.; mitral admitted 4 fingers.
68	Jessie K.	23	Mar. 15, 1894	„	Died, L.H., Sept. 8, 1897	Several times in hospital. P.M.—Adhesions; heart 32 oz.; mitral stenosis; large patent foramen ovale.
69	Annie S.	11	Mar. 21, 1894	„	Seen June 20, 1893	Has been laid up several times; dyspnœa and œdema of legs.
70	Alice Q.	16	May 10, 1894	„	Died, L.H., Aug. 29, 1895	Several times in hospital. P.M.—Adhesions; heart 27 oz.; myocarditis; mitral 3 fingers.
71	Eliza H.	8	June 29, 1894	„	Died, L.H., June 9, 1895	Several times in hospital; adhesions; heart 12½ oz.; mitral and aortic disease.
72	Ellen P.	14	July 4, 1894	?	Seen Oct. 5, 1898	Heart pulled over to right; retraction, &c.; poorly developed; often in hospital.
73	R se H.	8	Sept. 18, 1894	Rheumatism	Seen June 20, 1898	Wasted; irregular at school; adhesions diagnosed.
74	Grace D.	8	Oct. 21, 1894	„	Seen Aug. 7, 1898	Dyspnœa; pain; large heart; adhesions probable.
75	Florence H.	13	June, 1895	?	Seen June 24,	Often ill; at several hospitals; adhesions diagnosed.

No.	Name	Age	Date	Disease	Seen	Remarks
76	Ruth H.	13	1895	Rheumatism	Seen often Aug., Sept., 1898	Anæmic; wasted; large heart; double exocardial murmur at base.
77	Amy F.	10	Sept. 25, 1895	,,	Seen July 29, 1898	Frequently under treatment; large heart; exocardial at base; M.S. and doubtful M.P.S.
78	Ellen H.	6	Dec. 5, 1895	Chorea	Seen June 22, 1898	Only 5 weeks at school in 3 years; pain; wasting; large heart.
79	Phœbe H.	7	Dec. 5, 1895	Rheumatism	Died, St. Bart.'s Hosp., Sept., 1896	Adhesions diagnosed, confirmed by P.M. at St. Bartholomew's Hospital.
80	Florence D.	9	1896	,,	Seen Oct., 1898	Never well since; large heart; adhesions probable.
81	Violet H.	10	1896	,,	Seen July 10, 1898	Much dyspnœa and palpitation; large heart; M.S., M.P.S.; exocardial sounds.
82	Rose Le B.	20	April 8, 1896	Pneumonia	Seen July 10, 1898	Pain, dyspnœa, &c.; large heart; no bruit.
83	Rachel C.	7	June 18, 1896	Rheumatism	Seen often, 1898	In hospital 8 times in 18 months; large heart; adhesions.
84	Ellen T.	40	June 18, 1896	,,	Died at home, Sept., 1897	The attack was probably acute on old trouble. Died suddenly at home. Inquest: heart disease.
85	Eliza T.	18	July 29, 1896	,,	Seen June 18, 1898	No work; anæmia; dyspnœa; large heart; trouble chiefly aortic.
86	Maud B.	9	Aug. 9, 1896	Chorea	Seen June 18, 1898	Always ill; often in infirmary and hospital; large heart; adhesions diagnosed.
87	Clara C.	10	Aug. 19, 1896	Chorea; rheumatism	Died, L.H., April 12, 1898	Never well after; large heart: no P.M.
88	Ellen R.	4	Aug. 22, 1896	Rheumatism?	Seen June 13, 1898	Œdema from time to time; purpura; large heart; M.S.
89	Theresa M.	5	Oct. 11, 1896	Rheumatism	Seen Mar. 13, 1898	Often in hospital; very anæmic; large heart; adhesions probable.
90	Ellen C.	10	Nov. 13, 1896	,,	Seen Oct. 24, 1898	In hospital since with broken compensation; large heart; adhesions probable.
91	Caroline S.	8	Dec. 12, 1896	,,	Seen June 14, 1898	Very little school; in-patient since; large heart; adhesions probable.
92	Annie C.	14	Feb. 21, 1897	Chorea	Seen June 14, 1898	Anæmia; dyspnœa; heart area not much increased; M.S.
93	Priscilla E.	11	Feb. 24, 1897	,,	Seen May, 1898	In hospital, 1898, with broken compensation.

No.	Name.	Age.	Date of pericarditis.	Ætiology.	Latest information.	Remarks.
95	Annie B.	10	June 5, 1897	Rheumatism	Seen June 13, 1898	In hospital since; pain; large heart area; M.S.
96	Mary A. G.	17	June 29, 1897	,,	? Seen July, 1898	Admitted with great failure of compensation, May, 1898.
97	Alice G.	14	July 23, 1898	Rheumatism	Seen July 14, 1898	Compensation once broken since; heart area increased; M.S.
98	Eliza M.	8	Aug. 30, 1897	Chorea	Seen Oct. 13, 1898	Always under treatment as in- or out-patient.
99	Gertrude G.	8	Nov. 15, 1897	,,	Seen Nov., 1898	No school; large heart; adhesions diagnosed.
100	Annie W.	8	Nov. 23, 1897	,,	Died at home, Nov. 14, 1898	In hospital twice with broken compensation; no school; adhesions diagnosed; no P.M.

Cases where Compensation remains perfect.

MALES.

No.	Name.	Age.	Date of pericarditis.	Ætiology.	Latest information.	Remarks.
101	Walter H.	26	May 3, 1891	Rheumatism	Oct. 6, 1898	Lives in America; good health; information from brother.
102	William M.	19	Aug. 22, 1892	,,	Sept. 17, 1898	Light work; no symptoms.
103	William W.	21	Nov. 6, 1892	,,	,,	Engineer; rather anæmic; no symptoms.
104	Charles A.	27	Nov. 16, 1892	,,	Sept. 19, 1898	Builder's labourer; old aortic disease.
105	Charles T.	31	April 28, 1893	?	Aug. 10, 1898	Carman; no pain, but cannot carry very heavy weights.
106	Henry P.	23	Sept. 6, 1893	Rheumatism	Sept. 17, 1898	Waterside labourer; no symptoms.
107	George L.	14	Sept. 4, 1894	,,	Aug. 18, 1898	Sailing barge mate; curious attacks of pain.
108	John A.	36	Sept. 2, 1894	,,	June 22, 1898	Beer bottler; no symptoms.

No.	Name	Age	Date	syphilis	Date	A.C.D. increased,
110	Henry B.	18	May 30, 1895	Rheumatism	July 5, 1898	Grocer; cycles; no symptoms; signs of mitral stenosis.
111	William W.	27	March 26, 1896	,,	June 13, 1898	Brewer's labourer; no symptoms; soft M.S.
112	Arthur S.	33	Sept. 1, 1896	Renal	,,	Night coffee-stall keeper; slight dyspnœa.
113	David H.	32	Sept. 11, 1896	Rheumatism	,,	Cook; no symptoms.
114	Samuel T.	20	Sept. 21, 1896	,,	June 18, 1898	Shipwright; occasional pain at night.
115	Herbert B.	21	Oct. 10, 1896	,, chorea	Oct. 15, 1898	Railway work (G.E.R.); slight pain; initial attack very severe.
116	Woolf, H.	15	Oct. 30, 1896	Rheumatism	June 13, 1898	Bootmaker; occasional epistaxis; M.S.
117	Fred. R.	27	Nov. 7, 1896	Pleurisy	June 11, 1898	Glass-blower; no symptoms; exocardial sounds.
118	Alfred C.	16	March 13, 1897	Rheumatism	June 13, 1898	Works at lathe; occasional pain.
119	Samuel C.	19	April 10, 1897	,,	June 5, 1898	One attack of pain since.

FEMALES.

No.	Name	Age	Date	syphilis	Date	A.C.D. increased,
120	Sarah D.	35	Feb. 7, 1890	Rheumatism	Oct. 10, 1898	Well nourished; slight dyspnœa; M.S.; dyspepsia.
121	Alice M.	19	Nov. 12, 1890	,,	Oct. 4, 1898	Patient lying-in with third child; husband seen; general health good.
122	Ellen L.	14	Oct. 10, 1894	,,	July 21, 1898	In service; light place; no symptoms.
123	Annie T.	18	Oct. 10, 1895	,,	June, 1898	No symptoms; M.S.
124	Frances P.	14	April 21, 1895	,,	June 17, 1898	Tailoress; no symptoms; M.S.
125	Ellen M.	20	Aug. 10, 1896	,, ?	June 15, 1898	Letter from; "quite well."

No.	Name.	Age.	Date of pericarditis.	Ætiology.	Latest information.	Remarks.
126	Elizabeth B.	16	Nov. 12, 1896	Rheumatism	June 13, 1898	No symptoms; exocardial at base.
127	Annie C.	22	Dec. 11, 1896	,,	June 22, 1898	Married since; lives at Bristol; letter "quite well."

Three Cases in which death was primarily due to other causes.

No.	Name.	Age.	Date of pericarditis.	Ætiology.	Latest information.	Remarks.
128	Henry C.	28	June, 1898, Windsor Infirmary	Rheumatism	Died L.H., Nov. 25, 1895	Graves' disease and diabetes; death from coma; P.M. adhesions; heart 13 oz.; valves normal.
129	John B.	43	Christmas, 1894	?	Died L.H., May 16, 1896	Admitted with tachycardia; heart dilated; death from hæmatemesis; P.M. adhesions; heart 14 oz.; valves normal; gastric ulcer.
130	Fred. M.	37	March 31, 1896	Tubercle	Died at home, Sept., 1897	Tubercular pleurisy and pericarditis; later in hospital with phthisis, from which he died.

In these tables the following contractions are used :

A.C.D. = Area of cardiac dulness.
M.S. = Mitral systolic bruit.
M.P.S. = Mitral presystolic bruit.
A.S. = Aortic systolic bruit.

A.D. = Aortic diastolic bruit.
L.H. = London Hospital.
E.L.C.H. = East London Hospital for Children.

The dates given depend, except where otherwise stated, upon the Registers of the London Hospital and North-eastern Hospital for Children, or upon the writer's own investigations.

Note.—Case 14. This patient has since died. The pericardium was dilated and adherent. The heart weighed 36 oz. The valvular lesions were slight.

ON THE SPINAL ANIMAL

BEING THE

MARSHALL HALL PRIZE ADDRESS

BY

CHARLES S. SHERRINGTON,
M.A., M.D., F.R.S.

Read May 23rd, 1899.

MR. PRESIDENT AND GENTLEMEN,

When your Society was kind enough to recog
nise some work of mine by the coveted Marshall Hal
Prize, it extended to me at the same time your hos-
pitality for a short address. In answer to that invitatior
I now venture to lay before you a few remarks regarding
the physiology of the spinal cord. That the prize beques
to the Society is associated with the name of Marshal.
Hall sufficiently, I think, justifies my choice of such a
theme.

Marshall Hall, like not a few investigators of enthusiasm
seems to have been at little pains to know what of his
subject had been ascertained by previous workers. He
concluded from his own observations that the spinal cord
and the whole nervous system could be well regarded
from the physiological standpoint as a linked series of
reflex arcs. The idea was not a new one, but in his
hands it obtained new illustrative facts. The doctrine of
the cord as a spinal chain of functional segments the
units of which are reflex arcs, gained much furtherance
from the advocacy of Marshall Hall.

The segmental arrangement of the spinal nerves ir
their gross anatomy does not, of course, necessarily carry

with it any proof of the functional segmentation of the cord. That proof can be furnished only by analysis of the functional plan. A point of first importance for this question is the capacity of the fractionated spinal cord. Herbert Mayo [1] by his experiment showing the pupil reflex to be elicitable when but a single cranial segment remains, and Legallois [2] by his localisation of the respiratory centre in the bulb, had at the outset of the century laid the foundation of the segmental theory of the functions of the cord. Marshall Hall [3] and Grainger [4] further contributed experiments demonstrating the functional powers of spinal fractions. The latter drew significant observations from the reflex movements of invertebrata. The study of the functions of the nerve-chain of invertebrata became later neglected, most unwisely. Quite recently such work has vigorously been resumed. An example of its fruitfulness for the present problem is furnished by the observations of Hyde [5] on Limulus made in the Laboratory of Professor Loeb, of Chicago.

When in *Limulus* all that is analogous to the brain has been ablated, and indeed only the abdominal region of the spinal cord remains, the rhythmic respiratory movements of the abdominal segments still proceed regularly and co-ordinately. Even when a fraction of the nerve-cord, separated by transections in front and behind, is left corresponding with a single abdominal segment, the musculature of that segment continues its rhythmic action. Its rhythm is then no longer timed to that of the adjacent segments; its co-ordination with the rest is destroyed but its activity is maintained. Its activity ceases only if the segmental fraction of the spinal cord is itself destroyed. This instance is paralleled by the sexual " clasp " reflex of the brachial segments of the male frog, maintained when

[1] ' Physiol. Commentaries,' London, 1823.
[2] 1830.
[3] ' Memoirs on the Nervous System,' London, 1838.
[4] ' Functions of the Spinal Cord,' London, 1837.
[5] ' Journ. of Morphology,' 1894, vol. ix.

all the rest of the central nervous system is destroyed. Similarly in the cat and monkey, the reflex wagging o the tail persists when behind the spinal transection onl₁ the sacral region of the cord is left intact.

To judge how far the reactions of the spinal organ car be really considered as segmentally arranged, it is im· portant to have a conception as clear as possible of the spatial relations of the spinal nerve-cells. The delineatior of the spinal segment usually given presents its true extension very imperfectly.

The edifice of the whole nervous system is based, as upon two pillars, upon two nerve-cells, the afferent root· cell, and the efferent root-cell. These form a fundamenta. spinal arch upon which all other neural arcs are super· posed and functionally rest, immediately or mediately— even those of the hemispheral cortex. The afferent root· cells of the spinal axis may be arranged in three great groups : (1) cutaneous, from the sense organs of the skin; (2) " muscular," from the sense organs of the musculo· articular apparatus ; (3) " sympathetic," from the viscera. Each spinal afferent root consists typically of three con· stituent roots—a cutaneous, a muscular, and a visceral. The efferent root-cells are conveniently grouped in twc sets, one supplying skeletal muscles (1), the other entering the sympathetic chain (2) to innervate the musculature of the blood-vessels, of the skin, and of the viscera, includ· ing some secretory apparatus in the two latter.

To deal with the afferent root-cells first. The cutaneous afferent root-cells have their perikarya or cell bodies in the spinal ganglion. Probably in each one of the spinal ganglia the majority of the cells belong to nerve-fibres afferent from skin. The peripheral distribution of the col· lection of cutaneous nerve-cells of each spinal ganglion oc· cupies a semi-zonal field of body surface.[2] The zone is rela· tively wide and invests a little more than the entire width of one lateral half of the body, trespassing slightly across the

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[1] Goltz, 'Centralb. f. med. Wissensch.,' 1870.

[2] Sherrington, 'Phil. Trans.,' 1892, London.

middle line both ventrally and dorsally. In the regions of the trunk and neck and perinæum this zonal arrangement is quite obvious, but in the regions of the limbs it is less so, and at first sight appears departed from. The skin fields of the last three cervical and the first two thoracic and of the last two lumbar and the first two sacral ganglia are entirely confined to the limb and do not meet the middle line of the body either ventrally or dorsally. But when the skin fields of the ganglia of the limb[1] region had been carefully ascertained the results collated showed clearly that both in the brachial and in the pelvic limbs the zonal form of the fields still obtains, each semi-zone being wrapped half round the limb instead of half around the body. Hence exist what I have termed the *ventral* and *dorsal axial lines of the limbs*; these, forming, as it were, lines of watershed between the systems of semi-zones (Pl. XIII), meet at their one end the mid-ventral and mid-dorsal lines of the *body*, and may be looked upon as lateral extensions thereof. They are not hypothetical, for they are exhibited in the striping of animals. The dorsal axial line of the hind limb is particularly well seen in the tiger, the stripes in that limb starting from it; the dorsal axial line of the fore limb is in the same way well seen in the zebra. The dorsal axial lines of the fore limbs, diverging from the dorsal median line of the body, are in the ass usually marked by heavier pigmentation of the coat, thus making the "sign of the Cross" well known on the ass's shoulders. These axial lines of the limbs are of much clinical importance, for they are the boundaries observed by the upper limits of the anæsthesia accompanying injuries to the spinal cord or spinal roots in the regions of the lower and upper limbs respectively.

The skin fields belonging to the spinal ganglia are wide, and that for each ganglion largely overlaps its neighbours. In the monkey I have not been able to satisfy myself that there is any patch of skin in the neck, trunk, or limbs that is not in receipt of sensory supply

[1] Sherrington, 'Phil. Trans. Roy. Soc.,' London, 1892.

from each of two adjacent spinal ganglia. In certain regions (e. g. the hand, the foot, the pinna of the ear) the skin receives sensory fibres from each of *three* adjacent ganglia.[1] This explains how it is that the limb plexuses exist. The peripheral nerves of the limbs have to obtain components from more than one spinal nerve root. The innervation of the limb musculature is similarly plurisegmental. If, therefore, the spinal ganglion be considered a segmental collection of nerve-cells, those nerve-cells at their peripheral endings impinge on the body-surface over a zonal area which overlaps slightly with the contra-lateral zonal areas across the ventral and dorsal lines, and overlaps greatly with the collateral zonal areas next in front and next behind (headward and tailward) of itself. This zonal skin area may be considered a " segmental field " of skin.

As to which of the cell-bodies in the spinal ganglion belong to the skin-fibres, the probability is that the size of the nerve-fibres is a guide to the size of the cells, and that the " skin " cells are neither the largest nor the majority of the very smallest; they are probably the majority of the medium-sized cells.

The visceral constituent of the spinal ganglion is probably in all the ganglia numerically the weakest. It can be considered as practically if not absolutely wanting in the cervical ganglia and in the ganglia of the lower lumbar nerves. In order to estimate what number of the cells in a thoracic ganglion belong to its afferent path from the viscera, all that is necessary is to divide the spinal roots afferent and efferent proximal to the ganglion, and subsequently, when time has been allowed for degeneration, to count the number of myelinate fibres remaining sound in the white ramus communicans. In this way I found the sensory ganglion of the tenth thoracic nerve of the cat contain some 130 visceral nerve-cells and since the fibres are small the cells are probably small also.

It is curiously difficult to initiate reflex reactions by

[1] Sherrington, ' Phil. Trans. Roy. Soc.,' London, 1892.

sensory stimuli applied to the viscera themselves. In his great essay on " The Parts that feel and the Parts that do not feel,"[1] Albert von Haller in the last century brought forward abundant evidence of the extraordinary violence of insult that may be inflicted on the viscera even in absence of all anæsthesia—his observations were a hundred years prior to the advent of anæsthetics—and yet fail to evoke of sensation any sign whatsoever. Certain visceral conditions are, however, well known to be characterised by extremity of pain—instance the pain of renal or of biliary colic. In order to examine the afferent channels from viscera, I have had recourse to vascular reflexes obtainable in anæsthetised animals, and find they can be strikingly well provoked by the injection of a little fluid into such passages as the ureter and bile-duct. The mechanical distension transiently produced by injecting a few cubic centimetres of saline solution into the bile-duct immediately and regularly evokes a marked rise of arterial pressure. Plates XIV and XV are instances of the reactions which occur, typical bulbo-spinal reflex actions implicating the vaso-motor system ; vaso-motor spasm of considerable intensity and often presenting a double maximum succeeds each irritation.

These reactions seem to offer a means of experimentally determining the exact paths along which nervous impulses travel from these viscera to the spinal axis. I hope shortly to finish the work I have commenced in that direction ; the results obtained confirm those published by Dr. Bradford on the kidney,[2] and by Dr. Head [3] on the ureter and other viscera ; from the liver the afferent path as regards nerve-roots seems especially wide. Regarding overlap of distribution in the viscera neurons lying in adjacent spinal ganglia, little or nothing is known. Of the three great sets of channels, *vagus, thoracic afferent roots*, and *sacral afferent roots*, it is probable that at their

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[1] ' Opera Minora,' vol. i, Lausanne, 1772.
[2] Foster's ' Journal of Physiol.,' vol. xi, 1891.
[3] ' Brain,' 1893.

boundaries there is some overlapping in their peripheral distribution.

It has by a number of authorities[1] been denied that the skeletal muscles possess afferent nerves, but analysis of the nerve-trunks, entering and supplying the muscles, into their component fibres derived from the ventral and dorsal spinal nerve-roots respectively, demonstrates that the skeletal muscles receive quite a large number of nerve-fibres that are sensory. These nerve-fibres vary much in size, and some of them are the largest afferent nerve-fibres in the body. Many of them are, however, very minute, and these last seem to end in relation with the blood-vessels of the muscles. The sense-organs in the skeletal muscles are the "muscle-spindles," the tendon organs of Golgi, the end-bulb-like bodies in peri-neurium, the end-organs placed at junction of muscle-fibre and tendon especially exemplified in the eye muscles, and a few Pacinian corpuscles. In the nerves to some muscles the afferent fibres are as numerous as the efferent. It is probable that the very largest cells in the spinal ganglia belong to some of the nerve-fibres of the muscle-spindles. Probably in every spinal ganglion a number of the nerve-cells belong to the sense organs of muscle.

The topographical distribution of the afferent neurons of the skeletal muscles at the periphery will be best dealt with after dealing with that of the motor root-cells them-selves, and these will now be taken.

Regarding the efferent root-cells of the spinal cord, these, like the afferent, are divisible into three groups—those related to the skeletal musculature, to the skin, and to the viscera. The position of the nerve-cell bodies or perikarya of these efferent neurons, unlike that of the afferent, is intra-spinal. There is good evidence that they lie embedded in the cord at the same segmental level as the point of emergence from the cord of the nerve-fibres

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[1] v. Haller, Schiff, Kühne, &c.

they originate.[1]   If into the cord a clean incision be made transversely to its length, there ensues degeneration of the motor root fibres immediately at the site of the trauma and not in front of or behind that level; the root-fibres therefore do not take their origin any distance in front of or behind their point of exit or they would suffer degeneration.   This is well seen in regions where each motor root consists of a series of rootlets.   It proves each rootlet to be a collection of fibres which represents the nerve-cells lying in its own particular level of the grey matter, in fact, so to say, which drains only one particular cross-level of the cord.   This fact can be combined with the further observation that each constituent natural rootlet of the motor root contains fibres which, broadly speaking, are distributed to all the structures which the entire root innervates.[2]   Each rootlet of the root can thus be described as representing in miniature the entire root.   It follows that the position of the nerve-cells sending motor fibres to any one skeletal muscle is a scattered one, extending throughout the whole length of the spinal segments innervating that muscle; in the limb regions many muscles receive their motor fibres from as many as three consecutive spinal roots, and the bodies of the nerve-cells innervating those must therefore, inside the cord, extend through the length of three whole segments of the cord as a continuous columnar group, and in each transverse level of the cord these cells must lie commingled with nerve-cells innervating many other muscles. Hence no traumatic injury of the spinal cord can ever paralyse a single muscle alone and apart from others. Even the severance of any one whole motor nerve-root cannot paralyse a single limb muscle; the effect of such an injury is to partially impair a large number of the muscles.[3]

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[1] Sherrington, ' Journ. of Physiol.,' vol. xiv, 1892.   A. S. F. Grünbaum, ibid., vol. xvi, 1894.

[2] Sherrington, ' Journ. of Physiol.,' vol. xiii, 1892.

[3] Sherrington, ibid.

Analysis of the spinal nerve-supply of the muscles of either limb demonstrates that the muscular tissue of the limb is arranged in a number of rays,[1] there being one ray for each one metamer contributing to the limb. Of these rays the tailmost in the fore-and-aft series are the longest ; they extend to the extreme free apex of the limb, whereas the foremost, the most rostral, pass only as far as the thigh, the next hindward as far as the knee, the next hindward as far as the ankle. In the fore limb of *Macacus rhesus*, the common *rhesus* monkey, the four hind-most, most aboral rays all contribute to the musculature of the hand. When we inquire how these units of the segmental architecture of the limbs, these muscular rays, are related to the physiological or functional units of the limb musculature, it is at once obvious that the extent and boundaries of the two do not coincide. The definitely-bounded, individual and circumscribed masses of muscular tissue which are known as " the muscles " of the limb are functional elements of its structure as a physiological machine. But each of these functional elements is compounded and pieced together out of several rays or myotoms. Moreover, the boundaries between the myotoms do not correspond with the intervals, between muscles nor even with those between muscle-groups. Degeneration experiments which enable one to follow the distribution of the individual nerve-fibres of a root show that in some muscles the number of motor nerve-fibres given by a spinal root to a muscle is too small to evoke from the muscle any contraction obvious to inspection, for cases occur where a limb-muscle receives three, four, or five motor nerve-fibres from a particular nerve-root. This I regard as strong testimony to the morphological character of the overlap.[2]

Another feature of the distribution of the motor fibres of the spinal root to a muscle is the remarkable fre-

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[1] Sherrington, ' Journ. of Physiol.,' vol. xiii, 1892.

[2] Sherrington, Presidential Address to the Biological Society of Liverpool, 1898.

quency with which it is subject to slight individual variation. In examining a series of individuals (cats, monkeys) it is almost rare to meet two consecutive members of the series in which the root distribution is not by the degeneration or experimental method demonstrably somewhat different. Thus as instance I found in some individuals *supinator brevis* innervated from the sixth and fifth cervical nerves, in others, from the sixth and seventh. In the former case the innervation of the muscle may be termed "*prefixed*" type, in the latter "*post-fixed.*"[1] In my observations I considered it sufficient to group the individuals into two classes, a *post-fixed* and a *prefixed.* The *absolute* segmental level of a muscle is variable over the range of nearly a whole segment's length ; the *relative* segmental position is, however, preserved inviolably constant.

As regards the afferent nerve-fibres of the skeletal muscles, after their existence had been proved by the degeneration method, it was possible by the same method to examine their relation to the spinal segments. The result of such examination shows that not only are the skeletal muscles in many instances, pre-eminently in the limbs, plurisegmental as regards their motor innervation, but that they are so also in regard to afferent innervation. I was able to show[2] that the afferent nerve-fibres distributed to a given muscle arise in the root ganglia of exactly those spinal segments whence emerge the motor-fibres for the same muscle. In other words, the sensory nerve-cells directly connected with a given skeletal muscle are in any one individual always of the same segmental level as are the motor nerve-cells connected with the same given muscle. In so far, therefore, the simplest reflex arc connected with a muscle may be expected to lie exactly in those segments, whence issue the motor fibres of the muscle ; and is a segmental arc. In the "knee-jerk" we have evidence of a reflex arc traceable mainly *from*

[1] Sherrington, 'Journ. of Physiol.,' vol. xiii, 1892.
[2] 'Phil. Trans. Roy. Soc.,' London, 1896.

and *into vastus medialis* and adjacent part of *crureus*,[1] and this affords, as it were, a test case for the above conclusions. It confirms them perfectly. It exemplifies them by its narrow local extent, and by the segmentally horizontal correlation of the motor and sensory components.

What was said above to show that the intra-spinal site of the efferent root-cells is at the segmental level of the point of emergence of the motor cell-fibres itself applies to the motor nerve-cells of the sympathetic system, as well as to those of the skeletal muscles. The sympathetic efferent cells are not, however, present in all spinal segments, but are, as above noted, absent from the cervical and lower lumbar. They are distributed peripherally to the motor cells of the pre-vertebral ganglia of the sympathetic chain, and constitute the system of fibres which Langley[2] calls *pre-ganglionic*. Their distribution to the ganglia is an overlapping one, so that each pre-vertebral ganglion contains endings from a short series of sympathetic efferent spinal roots. Hence it is that so small a muscle as the iris receives efferent fibres from three spinal roots ; in the monkey the first, second, and third thoracic.[3]

It has by many been supposed that even in the cord of the higher vertebrata the collection of the perikarya of the efferent root-cells corresponding as they do with that of the afferent root-cells which form the spinal ganglia must be massed into a group for each spinal segment, separated by a distinct interval from that of the next segment.[4] As a fact, not only is evidence of such an arrangement as this wanting, but the evidence as regards the mammalian cord refutes the supposition.[5] So complete a fusion of the individual segments has in the course of ages gone on that, as regards the position of the efferent cells inside the cord, although no demonstrable degree of

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[1] Sherrington, ' Journ. of Physiol.,' xiii, 1892.
[2] ' Journ. of Physiol.,' xv, 1894.
[3] Sherrington, ' Journ. of Physiol.,' xiii, 1892.
[4] Schwalbe.
[5] Kaiser, v. Argutinski.

segmental interlap exists, yet also there is now existent no demonstrable segmental interval.

Just as the peripheral distribution of the afferent fibres of the spinal ganglia is an overlapping one, so also is their central intra-spinal ramification. The central or stem-process of each afferent root-cell, after entering the cord, bifurcates, as was discovered by Frithjoff Nansen,[1] the explorer. The headward branch of division ascends in the case of many fibres to the grey " nuclei" of the dorsal columns in the bulb; the aboral branch descends only a short distance, one or at most a few spinal segments. Each of these branches gives off collaterals into the grey matter, especially into the grey matter of the same segmental level as the root-ganglion, whence the root-fibre in question is itself derived. The intra-spinal extent of the afferent root-cell is therefore far more expanded than is that of the efferent root-cell. The dendrites of the latter seem to be practically confined to the segment in which the cell-body lies; the processes of the afferent fibres on the hand are traceable far into segments widely distant from that into which they plunge first on entering the cord. There is, therefore, a very extensive intra-spinal overlap in the central distribution of the afferent fibres derived from each afferent root-ganglion. Whether this applies equally to the afferent root-cells connected with the viscera as to other afferent root-cells is a question. The focal and circumscript character of the areas of tenderness and referred pain studied by Head in cases of visceral disorder, and their correspondence in situation with the skin areas of distribution of the individual spinal roots suggests that the central end-distribution of these visceral afferent fibres is less diffuse and less extensive intra-spinally and more concentrated within a single segment. On the limitations of intra-spinal root-overlap the study of the reference of visceral irritations may give most valuable light. Regarding the existence of segmental boundaries to the intra-spinal spread of

[1] Bergen's Museum, 1887.

impulses, observations such as the following illustrate th
slightness of the evidence in support. If the central en
of the afferent root of a thoracic nerve be carefully stimu
lated with gradually increasing strengths of stimulus
the musculature of the chest wall belonging to its ow:
motor root is the first to reply, and all parts of it do no
reply with equal readiness; certain portions are throw:
into contraction more readily than others. The *inter
costales* are rather late; as the intensity of the stimulus i
increased, the effect, curiously enough, is not easily pushe
across the median line; long before that passes, some o
the musculature supplied by the next adjacent motor root
behind and in front comes into play. It is difficult to not
a distinct step in the spread, and the spread passes ofte
a little earlier to the segment behind than to that in front.
Yet the intra-spinal resistance—to borrow electrical termi
nology—is for its own afferent path in the segment stimu
lated a little lower than in the adjoining. There is, there
fore, some functional segmentation. But in the case of th
limb muscles I could not obtain such evidence. Takin;
the flexion-adduction of the hallux of the monkey, thi
reflex could be obtained from each single one of a row o
the rootlets of the three last roots of the limb-plexu:
When obtained from an afferent rootlet of the first sacra
root it was obtained with equal facility, whichever of th
motor roots supplying the short muscles of the hallu
remained unsevered. There was thus no evidence of a seg
mental barrier of resistance between the spinal segment
concerned with the innervation of this muscle. Thes
segments in regard to this muscle seem to have becom
so welded together as to form a physiological unit.

There is, therefore, some evidence of functional seg
mentation in the cord; but such evidence is yielded bes
by the Invertebrata. In the Vertebrata it is an ances
tral heritage now so modified as to lie largely obscure
from recognition. It is partly on the assumption of
functional segmentation of the spinal cord that the custo

¹ Sherrington, ‘ Phil. Trans. Roy. Soc.,’ London, 1896.

mary manner of dealing with the spinal functions as a separate and integral chapter of physiology is justifiable. Otherwise the contents of that chapter would more properly be distributed under the various special chapters devoted to the separate senses and their organs and reactions. With Vertebrata this would, as a fact, for most purposes be the preferable plan.

The individual is a mass of living units, their activity co-ordinated together by conductive strands (nerve-cells) reacting to the environment. The environment acts on this co-ordinating system through "sense organs." Of these there are sets each attuned to certain species of environmental changes. For some changes in the environment no sense-organs have been evolved, *e.g.* for "Röntgen rays." The channels of access for the environment are olfactory, visual, auditory, gustatory, cutaneous, and, finally, the muscular and visceral. We might imagine the form of the individual and the disposition of the sense-organs as primitively very simple; for instance, a spheroid with a digestive cavity and sense-organs diffused over the surfaces especially the external. Such a form we should expect by evolution to become modified. Its contractile (muscular) mechanisms would obtain mechanical advantage (leverage) by elongation in certain directions. The lengthwise extension of the vertebrate body and of its lateral motor appendages, the limbs, are in so far such as might be argued *a priori*. Such extensions involve simultaneous extension of the covering surfaces; these surfaces are sentient, and the opportunity of these as sense-organs is almost in direct ratio as their mobility. The "touch-field" is thus enlarged, and becomes enhanced with greater "depth." Perceptions of space are favoured. The tactual sensations reinforce and check more efficiently those based on the eyes, and on the semicircular canals. The visual sense owes its preponderance in providing perception of space doubtless to its stimuli emanating from environmental sources relatively *remote*. Its reactions thus come to usually pre-

cede in time the other reactions occurring between th
organism and the environmental object. The brightness
or the darkness, or the colour affect the organism from
greater distance, and therefore, if the organism and objec
be relatively moving, in many cases earlier than do th
other qualities of the object. Sensations of the ey
in this way herald and forerun sensations that wil
in due time come to pass through other sense organs
Hence doubtless the almost constant relation betwee
direction of vision and direction of locomotion ; the tw
coincide. Progression habitually takes the organism int
that quarter of space already partly explored by hi
sense ; it carries him into his visual field. In th
majority of cases the visual organs are so set that thei
field of view is occluded as little as possible by the ex
tension of the organism itself. They are so set as to loo
" out" from some projecting portion of the contour c
the animal. When the form of the animal is elongate, a
it so often is, mechanical advantage of leverage havin
been thus acquired, the visual organs approximate 1
one of the poles. That pole will then " lead " in locom
tion—the eyes look " forward." In other words, th
motor mechanism that as it develops elongates, elongate
primarily " backwards." And it is covered with ski
possessing the usual cutaneous sense organs. And it ha
its own " muscular sense " due to organs embedded i
itself. Side appendages (limbs) thrown off laterally fro
the elongate trunk repeat the primary or axial elongatic
as regards mechanical leverage for motor elements and th
concomitance of increased sense apparatus of muscle ar
of increased sensorial skin surface. But, as a rule, th
viscera do not extend into the lateral appendages, althoug
for some distance into the primary axial or vertebrate, th
ensuring increased absorbent surface. Hence there come
to be a great motor apparatus extending back behind th
visual organ and containing besides muscle a certain amou
of visceral cavity ; and covered with skin containing cut
neous sense-organs. This apparatus it is which chief

executes the movements of the organism as a whole. It can alter the space-relations of the mouth and sense organs in regard to the environment and thus facilitate the nutrition of the organism by securing food; it can remove the individual from situations of danger or of injury. It is at the command not only of the sense organs resident in itself and in its own covering, but also of the great sense organs near that pole of the animal which its function is to drive "forward" into visual space. The pole where the great projicient senses of sight, smell, taste, hearing, and stereotropism (semicircular canals) have their organs is the pole that, by the action of the motor train attached, "leads" in progression, and the motor organ itself is after all in the main their instrument. As the animal scale is ascended it becomes their instrument more and more. The pole at which they lie is called by anatomists "the head," and the characters which the great sense organs and apparatus for intake of food impress upon it make its identification easy throughout a vast range of animal form. It is significant in the evolution of animal form that the organ that exhibits most uninterrupted and harmonious increase in development as studied successively in passing from lowest to highest is the brain. And it is significant that in the nervous system—segmental system as it is—the brain is developed not in those segments whose sense organs are ordinary cutaneous (tactual, &c.), muscular and visceral, but in the segments connected with the visual, olfactory, and otic sense organs; in other words, the brain is developed in the "head." The head is, so to say, the individual; it has the mouth, it takes the food, including air and water, and it has the main sense organs providing the data for both space and time. To this the body, an elongated motor organ with a share of the viscera and the skin, is appended primarily as a machine for locomotion. This latter must of necessity lie at behest of the great sense organs of the head.

Hence there are nervous conductors from the great sense-organs of the head to the great motor organ which

the contractile masses of the body together form. The
spinal cord contains this strand of conductors, and it is
in this sense a mere appendage of the brain. But the
motor organ itself is a complex structure, built up of many
parts. These, if the movements asked for by the projecting
senses are to be adequately executed, must be co-ordinate
in action. One of the main functions of the spinal cord
as an independent organ, and apart entirely from the
influence of the cranial sense organs, is the regulation of
the activities of the several muscles, so that they act co-
ordinately. This is well illustrated by analysis of the
muscular movements which ensue in the limbs as result
of reflex excitation in the purely spinal animal. In the
limb region, in response to an excitation of a single affe-
rent root, the spinal discharge of centrifugal impulses
evoked tends to occur by more than one efferent root.[1] If
is a plurisegmental discharge to plurisegmental muscles
But though plurisegmental it is in each segment only frac-
tional. It treats any one muscle of the limb as an entity
either exciting it wholly or leaving it wholly alone. The
contraction of the various segmental portions of the pluri-
segmental muscle are thus co-ordinated together. They
are knit together by a co-ordination which is wholly
spinal. Further, the co-ordination of antagonistic muscles
is managed by the spinal cord. After Winslow[2] and
Duchenne[3] it became current doctrine that antagonistic
muscles act co-operatively in the sense that the anta-
gonist by contracting synchronously with the prime muscle
modulates and helps to guide and control the nicety of the
movement. I have shown that in spinal and in bulbo
spinal reactions[4] (Pl. XVI, fig. 1), and also in many
cortical[5] (Pl. XVI, fig. 2), the reverse is in reality the con
dition of the antagonist. It is, in fact, relaxed, by inhibi

[1] Sherrington, 'Journ. of Physiol.,' xiii, 1892.

[2] 'Anatomical Expositions,' &c., 1749.

[3] 'Localisation,' &c., 1867, and Rieger, 'Archiv f. Psych.,' xiii.

[4] 'Journ. of Physiol.,' vol. xiii, 1892.

[5] 'Proc. Roy. Soc.,' vol. lii, 1893. See also Hering and Sherringtor
'Pflüger's Archiv,' 1897.

tion of its tonus and of any pre-existent contraction, at the same moment as the opposed muscle is by pressor influence thrown into contraction. For instance, when the motor neurons of the flexor muscles of the elbow are excited by pressor influences in a spinal reflex, the motor neurons for the extensor muscles are simultaneously inhibited as a part of the same spinal reflex (Pl. XVI, fig. 3). But not only are certain movements about a single joint opposed one to the other, certain movements at one joint are opposed to certain movements at neighbouring joints. Thus the extensors of the knee may be called antergetic not only to the flexors of the knee, but also to the flexors of the hip (Pl. XVII, fig. 1). In such cases the " reciprocal innervation," as I have ventured to style this mode of co-ordination, still holds good. The groups of motor neurons selected by the reflex action as it irradiates over spinal segments lying apart in the limb series are still those of synergetic muscles. For instance, while the reflex movement evoked by excitation of the fourth lumbar root or that responsive to the long saphenous nerve usually primarily contracts the flexors of the hip, it involves next not the antergetic muscles in the nearest spinal segments (*e. g. vasti* and *crureus*), but inhibits these and embouches into the synergetic of more distant segments, *e. g.* the hamstring muscles. In this way the reflex action, by its " spread," develops a combined movement—synthesises a harmony.[1] The mere inspection of a movement without further analysis of it is a very insecure guide toward judgment whether it be co-ordinate. The joints and muscles of the limb have been evolved contemporaneously and together in the course of the history of the individual and of the species. No muscle can therefore be thrown into action which will move the limb in any which is an unnatural direction. But from the above given criteria co-ordination is abundantly shown to result from the independent power of the spinal arcs, altogether apart from the influence of the

[1] ' Philos. Trans. Roy. Soc.,' 1896.

great cranial sense organs and of the cerebral arcs supei posed upon them. These senses and the brain fin elementary co-ordination of the skeletal musculature a achievement already provided and to hand in the spine cord itself. No doubt the product of the instrument is with the instrument itself, given over to their use in th reactions they elicit from the spinal musculature.

Now a co-ordinate movement must be " purposive " i character. Considered in the light of the Darwinia theory, the normal connections between any afferent pat (*e. g.* piece of skin) and the motor apparatus conjoine with it, *can* only be such as to lead to " purposive " move ment. The question rises, what are the purposes c which the reflex movements of a spinal animal are in dicative.

It is curious that the sense organs of both skin an muscle seem to evoke reflexly very much the same move ments the one as the other. Irritation of the skin of th foot evokes flexion at knee and hip ; so also does stretchin the muscular sense apparatus in the muscles and ligamenl at the pedal and ankle-joints. Certain reflexes are, how ever, peculiar to skin stimuli ; such are the " scratch reflex and the " shake" reflex in the spinal dog. Th former is started by stimuli applied to the skin of th trunk, perhaps especially of side, flank, and shoulder.] consists in a rhythmic flexion of the hind limb, causing it t execute scratching movement. The latter is started froi the skin of the trunk, especially perhaps along the mid dorsal line, and consists in a shake of the whole body like that given by a dog on coming wet from water. have seen it occur synchronously in the neck and hea above the lesion, and in the " spinal " part of th animal. Spinal transection had been performed at th sixth cervical level; on scratching the skin ove the scapula—partly anæsthetic, partly still sensitive— a " shake " was induced in the musculature above a well as behind the lesion. Grainger[1] concluded yeai

[1] 'Functions of the Spinal Cord,' London.

ago (1887) that spinal cutaneous reflex movements "are either of a preservative character or resembling the motions which the function of the organ requires." From the tegument of the spinal creature reflex movements of "preening" or "cleansing" can be elicited. The "preening" actions of the spinal insect and crustacean fall into this category. The spinal frog wipes irritants from its skin. The posturing of the hind limbs and tail of the spinal dog concurrently with reflex evacuation of the fæces, keeping the body from being soiled, come into the same class of purpose as the "shaking" and "scratching" reflexes above mentioned. The conjunctival reflex closure of the eye, essentially a cutaneous, and, from the broad point of view, a spinal reflex, is similarly preservative of the sensorial surface whence it is initiated.

Another purpose which seems unmistakably signified by spinal reflex movements is progression. To apply the appropriate stimulus for this is perhaps a little difficult. *Dytiscus*, the water-beetle, when reduced to a spinal condition does not run when placed on a firm surface, although each of its legs responds by free movement when individually stimulated. But on being placed in water it immediately swims [1] co-ordinately forwards. I find the spinal frog when placed in water at 38° C. swims for a short time co-ordinately forward with good bilateral strokes. Water at that temperature acts as a stimulus to the skin of the frog. The fish and triton after removal of the brain exhibit co-ordinate progression. In the dog, after spinal transection at the seventh cervical level, skin stimuli to the under surface of the body cause co-ordinate movements of all four limbs, the posture assumed taking the diagonal symmetry indicative of quadrupedal progression.[2] Even when the spinal transection is in the lumbar region, pressure upon the pad usually elicits protraction and flexion of hip and knee in the homonymous hind limb,

[1] Carpenter, 'Comparative Physiology,' London, 1850.
[2] Sherrington, 'Journ. of Physiol.,' xx, 1897.

and retraction with extension of hip and knee in th
crossed hind limb. The spinal reflexes significant (
progression seem to contribute chiefly toward preparator
posture in readiness for onset of action executed by th
musculature under the driving of higher centres. Thu
the well-known reflex spinal posture of the frog is flexio
of the hind limbs, the extensors of the joints being tau
and ready for the jump. Again with the primary refle
from the pedal end of the limb of the mammal. Thi
does not, as might have been expected, take the form c
the propelling stroke, the extensor push, that thrusts th
body forward. On the contrary, it lifts the limb fror
the ground. This is, no doubt, the phase of the limb'
movement of progression which requires the least outpu
of force; it resembles in so far the expiratory phas
preparatory for the inspiratory of respiratory movement
it may almost be likened to the diastole of the heart, anc
like both these, it is preparatory for a succeeding phas
of greater muscular effort. That phase ensues also in th
spinal animal, but it is the less easy to obtain and less soo
emerges from the depression of traumatic shock.

The spinal reflexes which in their results approximat
most closely to the normal reactions of the unmutilate
individual, are those connected with the pelvic an
abdominal viscera. Defæcation, micturition, parturitior
menstruation, genital turgor, seminal emission—these an
other analogous functions are executed as spinal reflex
in a manner presenting little or no physiological defec
from the normal. Their "purpose" is clear. If th
bulb be included with the spinal cord, and these togethei
including their peripheral nerves, be isolated from the res
of the nervous system, the animal as regards its viscers
life, including that of the heart and lungs, is practicall
intact. The viscus concerned with the intaking c
gaseous food is innervated from a cranial nerve, th
vagus; hence its "nervous centres" lie in the bull
An important part of the circulatory muscles, namel
the cardiac, is similarly innervated by a cranial nerve

hence likewise has a bulbar nervous centre. The existence of respiratory centres for the muscles of inspiration and expiration as independent from the bulbar centre remains still unproven. The existence of spinal vaso-motor centres subsidiary to the bulbar is asserted on the ground that it is still possible to obtain reflex alterations of blood pressure when the cord has been transected at *calamus scriptorius* if strychnia be exhibited to heighten its reflex activity. To demonstrate the existence of potent vaso-motor centres in the spinal cord does not, however, require the exhibition of strychnia. It is enough to allow the lapse of a few days or, better, weeks after transection of the cord in the cervical region. The blood pressure will then be found to have in the carotid of the dog a mean value of something over 100 mm. of mercury (Pl. XVII, fig. 2). By stimulating the skin mechanically or by temperature changes, or by faradising the central end of an afferent nerve, *e. g.* of the foot, reflex increase of blood pressure raising it 20 to 30 mm. of Hg is easily obtained. It is only in the first few hours immediately succeeding the initial trauma of transection that the cord being in a condition of shock gives no reflex response by its vascular musculature any more than by its skeletal musculature. This visceral shock seems no more severe in the higher than in the lower vertebrata. It is as regards the performances of the skeletal musculature that great difference exists in regard to shock distinguishing between the spinal frog and spinal monkey. I have ventured to suggest that the spinal shock of the latter animal is connected with an isolation dystrophy such as occurs in cases where nerve-cells habitually actuated by other nerve-cells are suddenly and completely cut off from their influence. That the difference is very great and real between lower and higher types in this respect of spinal shock is shown by such instances as the following. The cat from which the Rolandic area of the cortex has been removed, so as to ablate the whole of the limb centres from one hemisphere, if some weeks later " decerebrate rigidity " be induced in its limbs, yields the

rigidity without perceptible difference between the sides both right and left. But in the monkey similarly prepared a great difference between the limbs of the two sides is apparent. The rigidity on the side crossed to the cerebral lesion is very much less than on the homonymous side. At the same time it must not be thought that the whole depression of function in the parts innervated behind the spinal transection is due to removal from them of merely cerebral influence. That that cannot be the case is shown by a fact that I have several times had opportunity to observe, namely that the performance of a second spinal transection some weeks later, and some segments behind a former spinal transection, is followed by the recrudescence of many of the original symptoms of depression of function that had followed the original transection It is significant that such a second transection behind a previous one causes a considerable increase in the descending spinal degeneration, showing that there descend from upper parts of the spinal cord many channels arising in those upper spinal regions and connecting them with other spinal regions further back.

Whence comes the great difference existent between, on the one hand, ape and man, and, on the other, fish and frog, as regards the depression of the reactions of the skeletal musculature ensuent upon total transverse lesion of the spinal cord ?

To refer briefly for a moment to initial shock due to spinal section in the monkey, there can hardly be witnessed a more striking phenomenon in the whole physiology of the nervous system. From the limp limbs, even if the knee-jerks be elicitable, no responsive movement, beyond perhaps a feeble tremulous adduction or bending of the thumb or hallux, can be evoked even by insults of a character severe in the extreme. That which the delicate yellow spot is to the sensifacient sheet of the retina, may the thumb and index be said to constitute in the great sensifacient field of the limb. Nevertheless a hot iron

laid right across the thumb, index, and palm remains an absolutely impotent excitant, or able only to evoke a faint flexion of the thumb; the crushing of a finger has no greater effect. A huge afferent nerve, such as the internal saphenous, containing some five thousand sensory nerve-fibres, when laid across the electrodes and subjected to currents absolutely unbearable upon the tongue, elicits no response, and probably no movement whatsoever. To the whole popliteal nerve, representing an area of sentient skin which includes the entire sole and much of the leg besides, intolerable faradisation can be applied without response. A more impassable condition of block or torpor can hardly be imagined; its depth of negation resembles, to superficial examination, profound chloroform poisoning.

Shock is not only more severe in the monkey than in the other laboratory types, but it is also more lasting, and its symptoms are more profound and prolonged than in any other animal I have observed. The symptoms of shock, in many monkeys, persist for days instead of hours and minutes, as in cat and dog. It is important to note that in the monkey, much of what we are, from observations upon the lower animal types, inclined to regard as temporary, and relegate to block or "shock," in Goltz's language "*Hemmungserscheinungen*"—not "*Ausfallserscheinungen*"—proves, under prolonged observation to be, I must admit, permanent; in fact, to be *true deficiency phenomenon*. Every histologist acquainted with the comparative structure of the spinal cord in the ape and in the dog must have been impressed with the far greater complexity obvious in the former. The above evidence is in accord with that, for it shows that the same trauma inflicted upon the cord leads, in the monkey, to much heavier permanent defect than in the dog; just as, in fact, ablations of the cortex cerebri are pregnant with far greater "*Ausfallserscheinungen*" in the monkey (Ferrier, H. Munk, Schäfer, Mott) than in the dog (Goltz). It is reasonable to argue still severer results in the case of the human spinal cord;

of which, again, we know the minute structure to be yet more complex still. *The permanent damage done is there-fore, as well as the initial shock, disproportionately greater in monkey than in cat and dog.*

My own experience leads me to think that the condi-tion of a spinal cord isolated by a spinal transection is often more normal a few hours after the transection than it is when long periods of weeks and months are allowed to elapse. I am well aware that this is contrary to the opinion of Goltz and others. The advantage believed to accrue from waiting is that the phenomena of shock may have time to pass off as completely as possible. How long the phenomena of shock may last at longest is a question on which very different views are held. Goltz to whose trenchant observations and bold system of ex-periment we owe so much of our knowledge of the physiology of the central nervous system, is the founder of a school which works in the belief that the pheno-menon of shock may persist for months, even years. It is, as far as not, merely a matter of nomenclature, a question on which no definite decision seems as yet possible. I myself have gradually been driven to the belief that "shock" does not take long to pass off, *i. e* does not at longest persist for more than a few weeks I am not considering here the complications arising out of long badly-healing and suppurative wounds, and the continual irritation they may produce if situate in the nervous system. But though shock passes off, the alterations produced in the isolated cord or piece of cord (by permanent withdrawal of the influences it has lived accustomed to receive from other portions of the central nervous system) progress, and are in a sense cumu-lative. The decreasing depression merges—at present inextricably for us—in the increasing onset of an "isolation-dystrophy." Much of what is called "shock," in regard to the mammalian cord, is, I believe, due to "isolation-dystrophy," and is really permanent,—that is to say, would not pass away if the animal were to live

healthily for any number of years. The most favourable time for the examination of the independent capabilities of the spinal cord is that when the sum of "shock" and "isolation-dystrophy" together is of smallest amount. That time, compounded as it is of two such variable factors, is itself extraordinarily variable. In result of spinal transection in monkey, I am sure that "shock" lasts longer, and that "isolation-dystrophy" comes on earlier than in the other animal types commonly observed in the laboratory. It is the conjunction of the periods of these two phenomena which renders so difficult and so largely defeats attempts at observations on proper spinal reactions of the monkey. If the overlap of the two is great, then no spinal reflexes, or only the merest traces of them, may be observable. In man it is only natural to suppose—and what clinical experience I have had access to strengthens me in the belief—that even more than in monkey will "shock" be protracted, and "isolation-dystrophy" speedy and severe. The observations of Bastian,[1] Bowlby,[2] and Bruns,[3] teach us that the clinical picture of the effects of total transverse lesions of the human spinal cord does not accord in the way that medical text-books have been wont to describe with the long known results obtained from the transected cord by the physiologist. Older physiological experiments are, however, not based on nervous systems so approximate to the human as is that of Macacus, Cercocebus, &c. Of these latter I would say that their condition after spinal transection commonly resembles in its features in the most striking manner the condition of spinal depression observed after spinal translesion in man, and considered by Bastian to be the typical status.

My results on monkeys bear out strikingly and fully what Bastian describes as the typical condition in man, after complete transverse destruction across the cord.

[1] ' Medico-Chirurgical Transactions,' London, 1891.
[2] Ibid.
[3] ' Neurologisches Centralblatt,' Berlin, 1893.

The chief difference is that in the monkey in most case
—partly, perhaps, because the lesion is more localised b;
experimental infliction than by accidental—the depressioi
is not so severe. For instance, the knee-jerk, which dis
appears almost immediately after the transection, return
usually in a week or ten days,[1]—often, however, not fo
three weeks; occasionally, on the other hand, in tei
minutes.

The great motor organ—the skeletal musculature—i
at the command of the sense-organs. Not only is i
actuated by contact sensations evoked in the neural systen
of the individual by the tangible quality of the circum
ambient environment; each light that causes the anima
to move, each sound, each odour, shows how the moto
machine lies at the behest of the great sense-organs o
the head. Now these latter are broadly distinguishabl
from the sense-organs of the trunk inasmuch as they sub
serve sense possessing the *quale* of " projection." Fo
each individual creature the material universe is thus sepa
rated into two parts, the part that is " me," and the par
that is " not me." I think it was Lotze who said tha
doubtless to the trodden worm, of these two halves th
trodden " me " shall surely appear the greater. By
high spinal transection the splendid motor machinery o
the vertebrate is practically as a whole and at one strok
severed from all the universe except that fraction th
" material me." The deeper depression of reaction int
which the higher animal as contrasted with the lower sink
when made " spinal," appears to me significant of thi
that in the higher types, more than in the lower, the grea
projecting senses actuate the motor organ, and impel th
motions of the individual. That deeper depression show
how, as the individual ascends the scale of being, the mor
percipient, the more cognizant does it become of th
circumambient universe outside that is " not me;" an
thus the latter acquires a more and more preponderar
directive influence over those reflections, those expression
of the creature's neural states, its " doings."

[1] 'Foster's Journal of Physiology,' vol. xiii, 1892.

DESCRIPTION OF PLATES XIII—XVII

Illustrating The Spinal Animal (Charles S. Sherrington, M.A., M.D., F.R.S.).

Plate XIII

Fig. 1.—This figure indicates the position of the limits of the skin-fields of the cervical and brachial spinal nerve-roots. Both the anterior and the posterior borders of the fields are indicated in the diagram, which renders the figure somewhat confusing. A portion of the limit of each field is, however, omitted, namely, that portion of it which joins the portions of the adjacent fields to form the *ventral axial line* of the limb. This combined portion of the borders forming the ventral axial line of the limb is represented by itself in fig. 2 of this same Plate XIII. The lines which in fig. 1 indicate the *posterior* limits of the fields are finely dotted lines, thus ⋯⋯⋯⋯; the lines which indicate the *anterior* limits of the fields are simply broken, thus — — —. The number attached to each line signifies the number of the cervical or thoracic nerve-root in the enumeration of the vertebral series; thus 4 — — — signifies the position of the anterior limit of the skin-field belonging to the fourth cervical nerve-root, and 1 . . . . signifies the position of the posterior limit of the skin-field belonging to the first thoracic nerve-root. The line v signifies the posterior border of skin-field of the cranial fifth nerve.

Fig. 2 shows the position of the *ventral axial line of the brachial limb*, v. a. l., as formed by the conjunction along the place indicated of the borders of the consecutive skin-fields of the brachial nerve-roots.

Figs. 3 and 4.—These figures indicate in the same way as the preceding the positions of the limits of the skin-fields of the cervical and brachial nerve-roots, and how from those limits an *axial line of the limb* is revealed by their conjunction along a certain line. These figures differ from the two preceding only in giving the dorsal aspect of the limb instead of the ventral, d. a. l., the *dorsal axial line*. In fig. 3 the line v signifies the posterior limit of the fifth cranial nerve.

Plate XIV

b. p. = the carotid blood-pressure in a cat (chloroform and ether, in addition to morphia and curare). An injection of 2½ cubic centimetres of normal saline solution at body temperature was made into the common bile-duct at the place marked by the signal. A marked rise of the carotid pressure ensued. The time is marked in seconds.

PLATE XV

B. P. = blood-pressure tracing from the carotid artery of the cat. S. Electric signal. T. Time marked in seconds. From 1 to 0 on the signal trace 1 cubic centimetre of normal saline solution at body temperature was introduced from a reservoir into the left ureter, about five seconds. The animal was chemically narcotised with a mixture of chloroform and ether and by a hypodermic injection of morphia : it was paralysed with curare Tracing reads from left to right.

PLATE XVI

FIG. 1.—Reciprocal innervation at the elbows of the monkey, both reacting under one spinal reflex. Flexion of one elbow is seen to accompany extension at the other, and at both reciprocal innervation of the antagonistic muscles is evident.

FIG. 2.—Reciprocal innervation of the lateral muscles of the eyeballs under cortical excitation of the left hemisphere, producing consensual movement of the eyeballs to the right, the sixth nerve of the right side having been severed at its exit from the base of the brain. The right eyeball is shown to move to the right up to the median position ; the left to beyond that.

FIG. 3.—Reciprocal innervation instanced by the extensors and flexors of the elbow-joint in the monkey, obtained as a reflex from the twig of the radial nerve, the animal being in the condition of "decerebrate rigidity." The tonic contraction of the extensors relaxes as the reflex contraction of the flexors sets in.

PLATE XVII

FIG. 1.—Similar to the above, but obtained from the flexors and extensors of the knee-joint, and with levers, writing not in harmonious sense as above but in opposing sense, so that the relaxation of the lower rectus femoris muscle is written upwards.

FIG. 2.—B. P. Graphic record of the arterial blood-pressure of a dog (carotid artery) under chloroform and ether after morphia, and in addition to curare The spinal cord had been completely severed transversely at the seventh cervical segment eight weeks prior to the observation. The gap in the signal trace shows when, for five seconds, the toes of one hind foot were forcibly extended. The movement caused a reflex rise in the arterial blood-pressure, followed by a temporary fall. Zero of B.P. marked the level of the float in the manometer under atmospheric pressure. It is noteworthy that there is acceleration of pulse as well as rise of the arterial pressure.
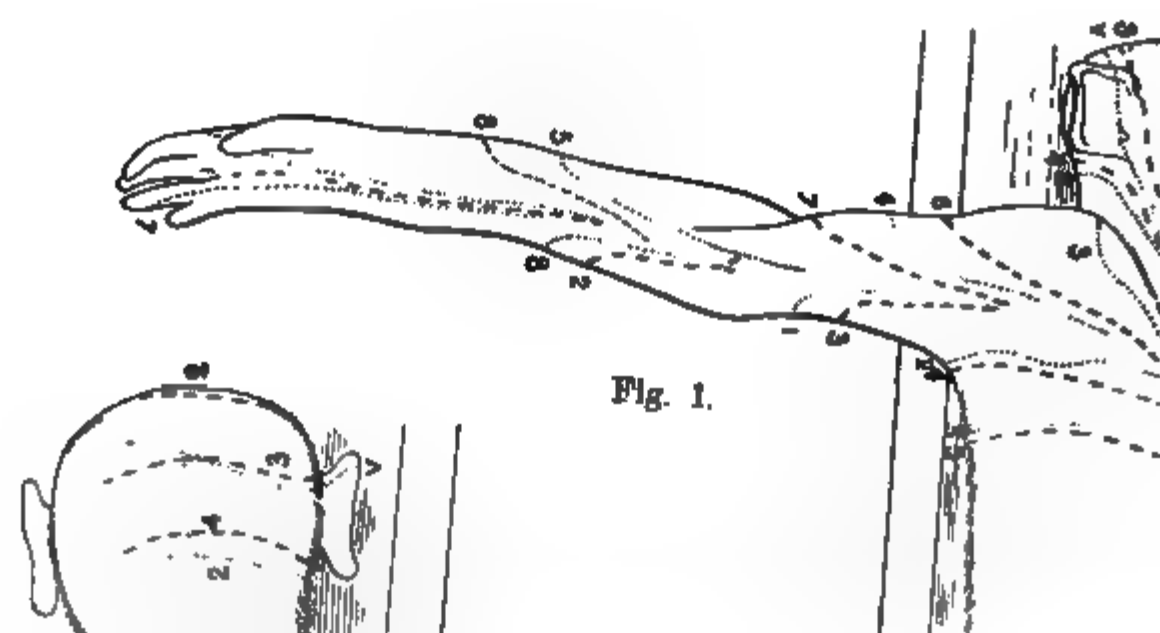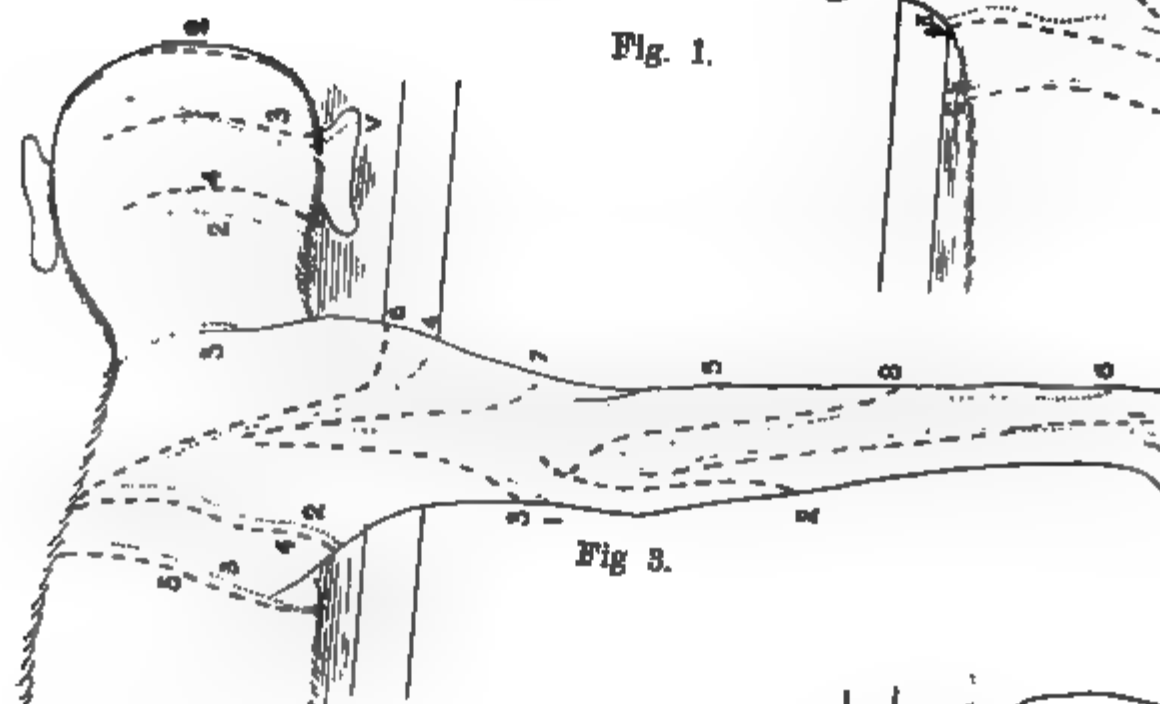
Plate XIII.

Med. Chir. Trans., Vol

Fig. 1.

Fig 3.

Fig. 2.

D.A.L.

Fig. 4.

Plate XIV.   Med.-Chir. Trans , Vol. LXXXII.

B. P.

Signal.

Time.

Plate XV.   Med.-Chir. Trans., Vol. LXXXII.

B. P.

S.

T.

Plate XVI.   Med.-Chir. Trans., Vol. LXXXII.

Fig. 1.

Fig. 2.

Fig. 3.

Plate XVII.   Med -Chir. Trans., Vol. LXXXII.

Expt. 31
18th Nov. 1894

Semimemb

Rect. fem

FIG. 1.

B. P

Zero of B. P.
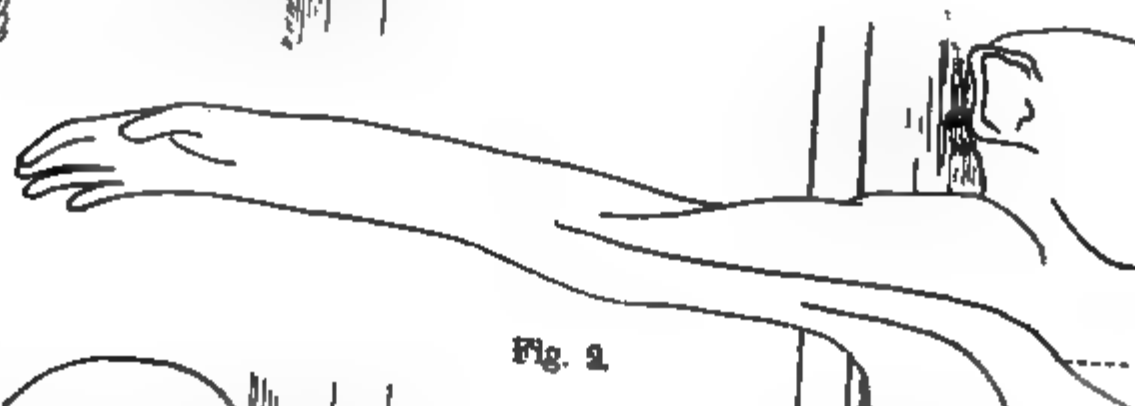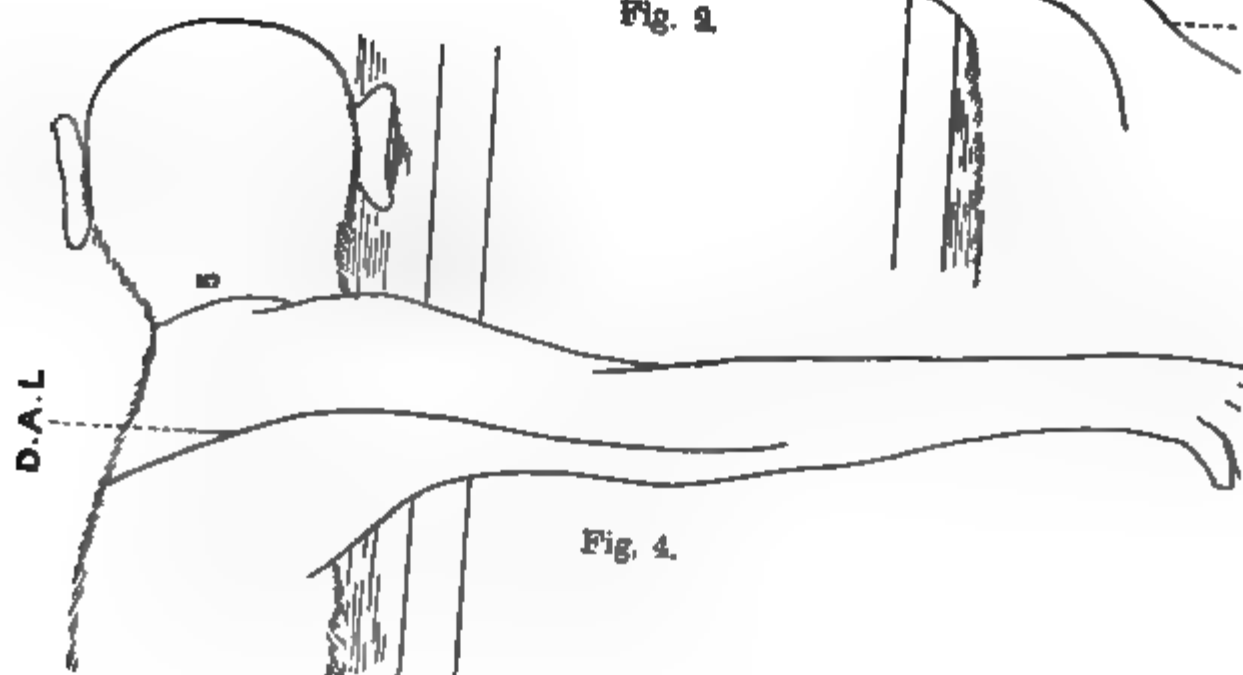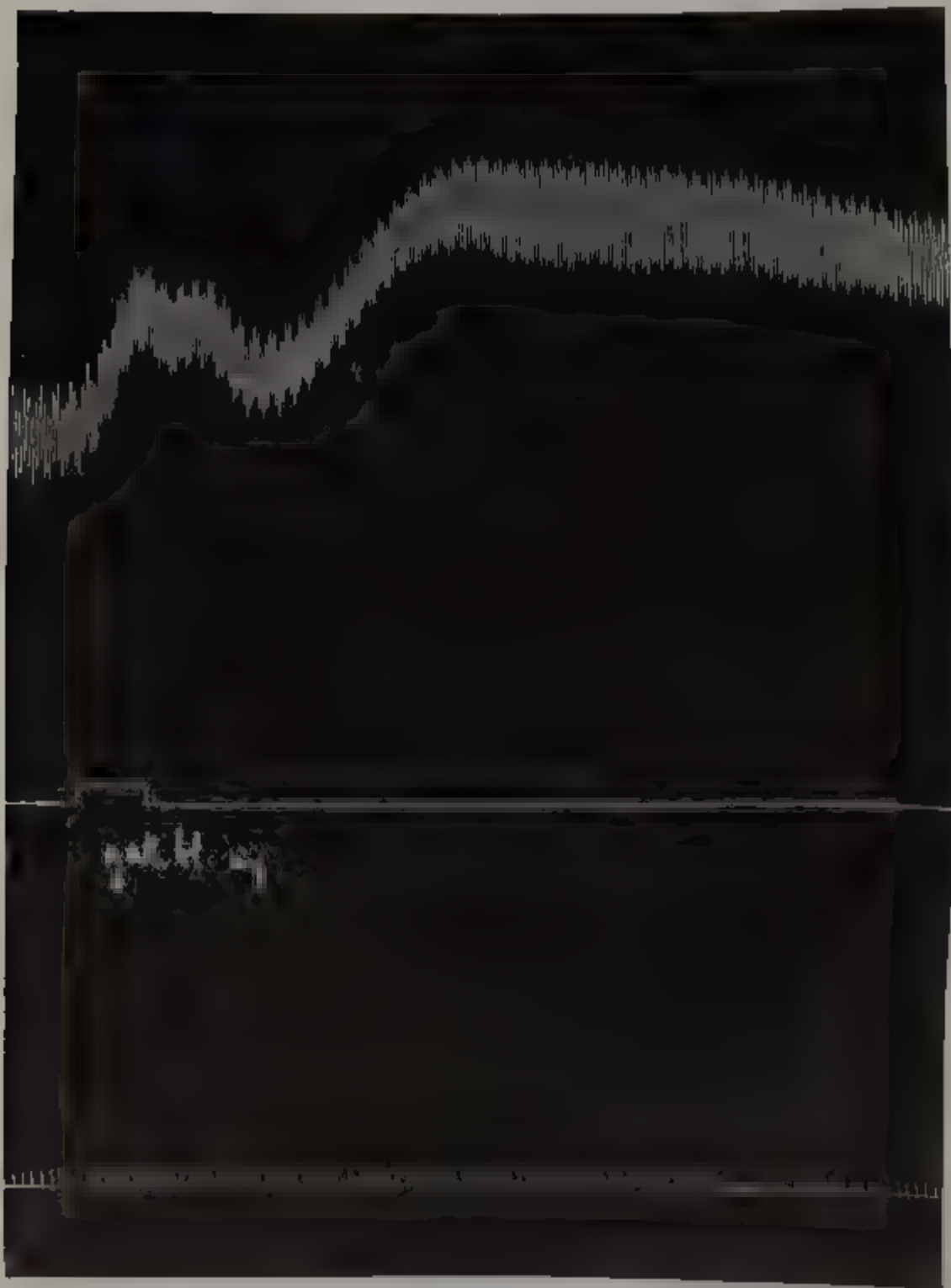
Signal.

Time.
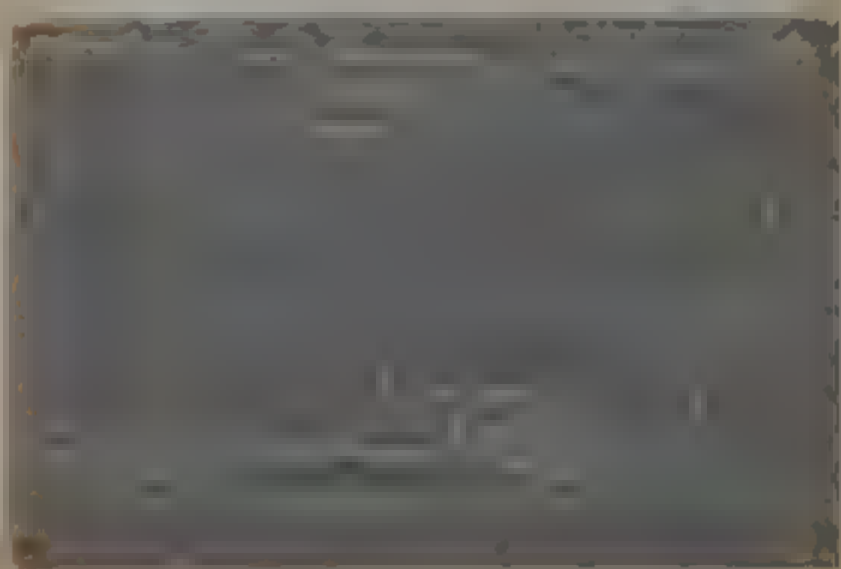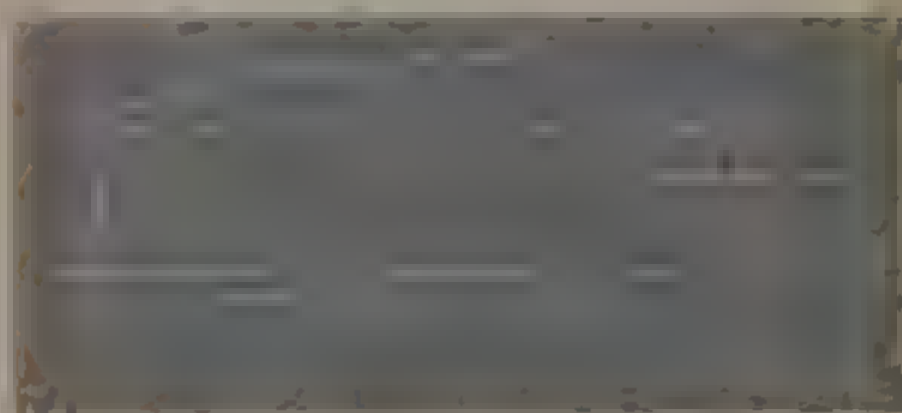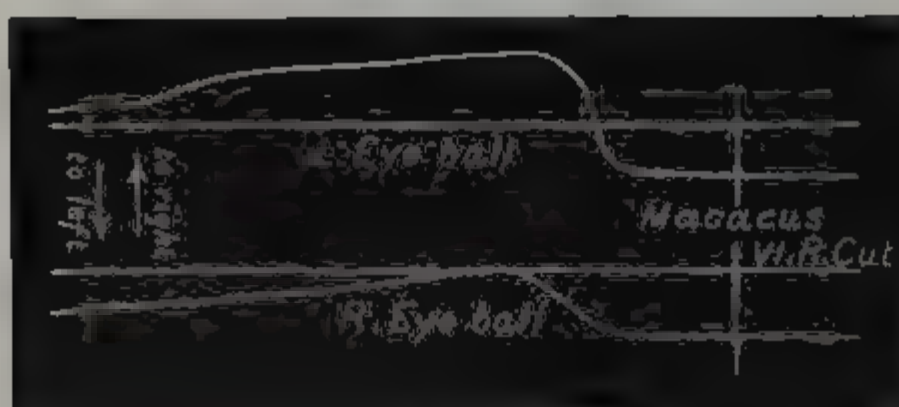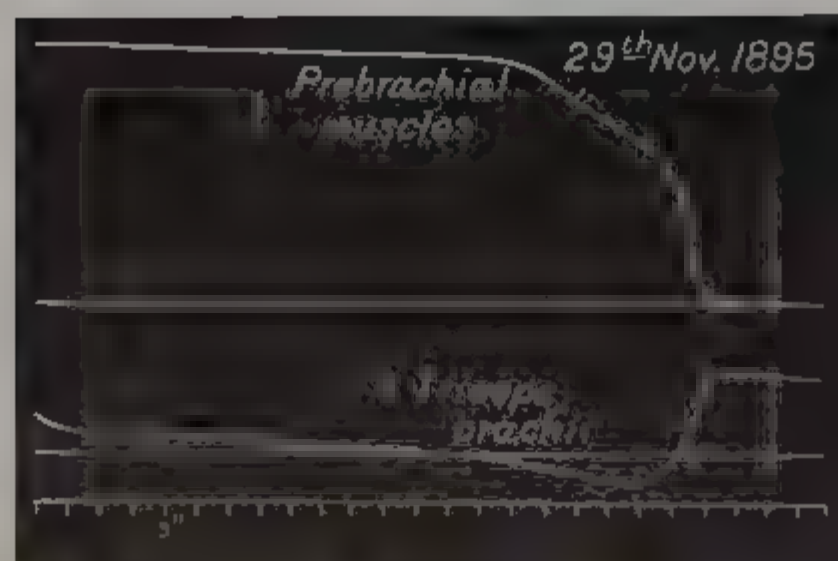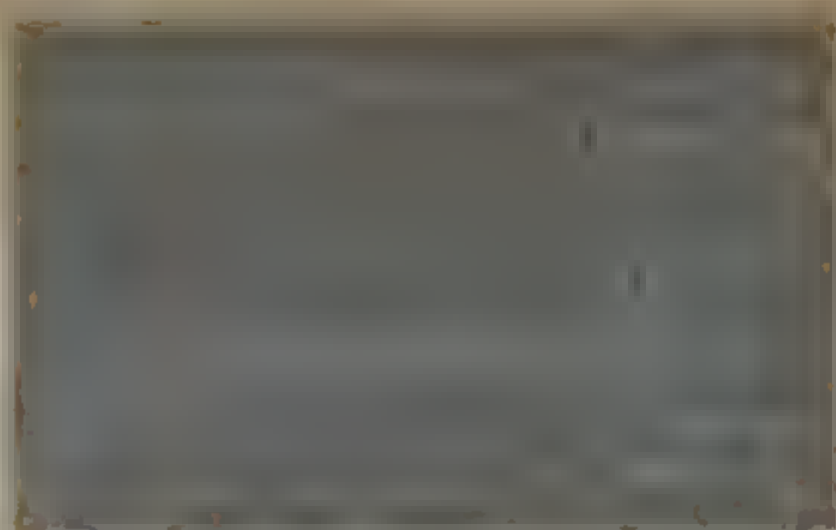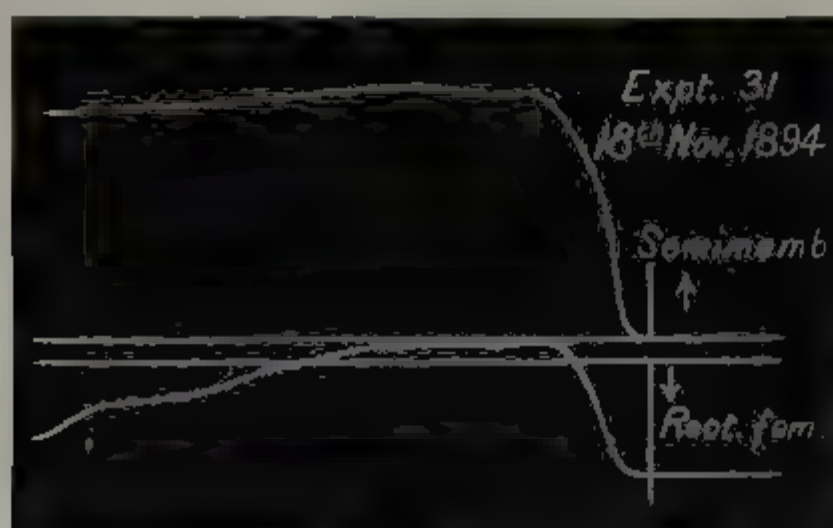
FIG. 2.
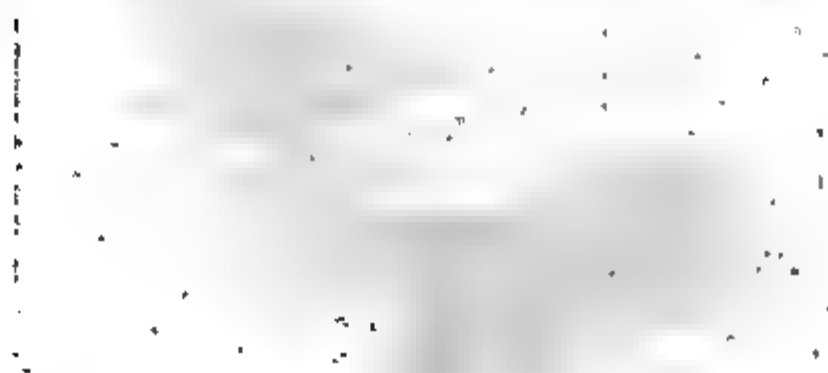
INDEX

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